U.S. DEPARTMENT OF COMMERCE
National Technical Information Service

PB-256 734

Air Pollution Assessment of Benzene

Mitre Corporation

Prepared For
Environmental Protection Agency

April 1976

BEST SELLERS

FROM
NATIONAL
TECHNICAL
INFORMATION
SERVICE

Comparison of Energy Consumption Between West Germany and the United States
PB-245 652/ PAT 112 p. PC$5.50/MF $3.00

A Comprehensive Policy to Ameliorate Adverse Impacts of Transportation Facilities
PB-247 823/ PAT 138 p. PC$6.00/MF $3.00

A Comprehensive Policy to Ameliorate Adverse Impacts of Transporation Facilities, Appendix
PB-247 824/ PAT 310 p. PC$9.75/MF $3.00

Demand for Coal for Electricity Generation 1975–1984
PB-245 216/ PAT 40 p. PC$4.00/MF $3.00

A National Study of Adolescent Drinking Behavior, Attitudes and Correlates
PB-246 002/ PAT 405 p. PC$11.00/MF $3.00

Energy from Coal: Guidelines for the Preparation of Environmental Impact Statements
PB-242 960/ PAT 258 p. PC$9.00/MF $3.00

Energy Versus the Environment: The Issues
PB-246 382/ PAT 35 p. PC$4.00/MF $3.00

Improving the Dissemination of Scientific and Technical Information: A Practitioner's Guide to Innovation
PB-247 057/ PAT 203 p. PC$7.75/MF $3.00

Investigation and Evaluation of Cracking in Austenitic Stainless Steel Piping of Boiling Water Reactor Plants
PB-246 645/ PAT 123 p. PC$5.50/MF $3.00

Pipeline Industry's Practices Using Plastic Pipe in Gas Pipeline Facilities and the Resulting Safety Factors
PB-247 301/ PAT 436 p. PC$11.75/MF $3.00

NOx Combustion Control Methods and Costs for Stationary Sources. Summary Study.
PB-246 750/ PAT 117 p. PC$5.50/MF$3.00

Reactor Safety Study: An Assessment of Accident Risks in U.S. Commercial Nuclear Power Plants
PB-248 200/SET/ PAT 2,504 p. 8 Volumes PC$61.00

Report on Sulfur Oxide Control Technology
PB-246 867/ PAT 89 p. PC$5.00/MF$3.00

Research Evaluation of a System of Natural Air Conditioning
PB-243 498/ PAT 384 p. PC$10.50/MF$3.00

Retrofitting a Residence for Solar Heating and Cooling: The Design and Construction of the System
PB-247 482/ PAT 101 p. PC$5.50/MF$3.00

Saudi Arabia Five-Year Plan, 1975–1980
PB-246 572/ PAT 683 p. PC$16.25/MF$3.00

Shotcrete Practice in Underground Construction
PB-248 765/ PAT 501 p. PC$12.75/MF$3.00

Short-Term Coal Forecast, 1975–1980
PB-247 073/ PAT 120 p. PC$5.50/MF$3.00

State Energy Information Systems: A Survey
PB-247 457/ PAT 64 p. PC$4.50/MF$3.00

A Study of Fixed Capital Requirements of the U.S. Business Economy
PB-248 690/ PAT 84 p. PC$5.00/MF $3.00

Sulfates and the Environment—A Review
PB-248 122/ PAT 155 p. PC$6.75/MF $3.00

HOW TO ORDER

When you indicate the method of payment, please note if a purchase order is not accompanied by payment, you will be billed an addition $5.00 *ship and bill* charge. And please include the card expiration date when using American Express.

Normal delivery time takes three to five weeks. It is vital that you order by number or your order will be manually filled, insuring a delay. You can opt for *airmail delivery* for a $2.00 charge per item. Just check the *Airmail Service* box. If you're really pressed for time, call the NTIS Rush Order Service. (703) 557-4700. For a $10.00 charge per item, your order will be airmailed within 48 hours. Or, you can pick up your order in the Washington Information Center & Bookstore or at our Springfield Operations Center within 24 hours for a $6.00 per item charge.

You may also place your order by telephone or TELEX. The order desk number is (703) 557-4650 and the TELEX number is 89-9405.

Whenever a foreign sales price is NOT specified in the listings, all foreign buyers must add the following charges to each order: $2.50 for each paper copy; $1.50 for each microfiche; and $10.00 for each Published Search.

Thank you for your interest in NTIS. We appreciate your order.

METHOD OF PAYMENT

☐ Charge my NTIS deposit account no._____

☐ Purchase order no._____

☐ Check enclosed for $_____

☐ Charge to my American Express Card account number

Card expiration date_____

Signature_____

☐ Airmail Services requested

NAME_____

ADDRESS_____

CITY, STATE, ZIP_____

Item Number	Quantity		Unit Price*	Total Price*
	Paper Copy (PC)	Microfiche (MF)		

All Prices Subject to Change 8/76

Sub Total	
Additional Charge	
Enter Grand Total	

Clip and mail to:

NTIS

National Technical Information Service
U.S. DEPARTMENT OF COMMERCE
Springfield, Va. 22161
(703) 557-4650 TELEX 89-9405

PB 256 734

MTR-7188

Air Pollution Assessment of Benzene

P. WALKER

APRIL 1976

REPRODUCED BY
NATIONAL TECHNICAL
INFORMATION SERVICE
U. S. DEPARTMENT OF COMMERCE
SPRINGFIELD, VA. 22161

MITRE

TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completing)

1. REPORT NO.	2	3. RECIPIENT'S ACCESSION NO.
MTR-7188		

4. TITLE AND SUBTITLE	5. REPORT DATE
Air Pollution Assessment of Benzene	April, 1976
	6. PERFORMING ORGANIZATION CODE
7. AUTHOR(S)	8. PERFORMING ORGANIZATION REPORT NO.
P. Walker	
9. PERFORMING ORGANIZATION NAME AND ADDRESS	10. PROGRAM ELEMENT NO.
The Mitre Corporation McLean, Virginia 22101	11. CONTRACT/GRANT NO. 68-02-1495
12. SPONSORING AGENCY NAME AND ADDRESS	13. TYPE OF REPORT AND PERIOD COVERED
U. S. Environmental Protection Agency Office of Air and Waste Management Office of Air Quality Planning and Standards Research Triangle Park, North Carolina 27711	Final 14. SPONSORING AGENCY CODE EPA-AWM

15. SUPPLEMENTARY NOTES

16. ABSTRACT

Benzene acts primarily as a central nervous system depressant although it has also been shown to cause hematopoietic dysfunction and myelotoxicity in humans and animals. Production of benzene in the U. S. can utilize both coal and petroleum; however, the majority of the production is petroleum derived. Exposure of the human population to benzene can ensue not only from production facility emissions, but from usage of the compound, from oil spills, gasoline evaporation, and coke oven emissions. Control technology has been developed which, if utilized, can provide protection to industrial workers.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
Organic Compounds Hydrocarbons Organic Solvents Physiological Effects Air Pollution Control Stationary Sources	Air Pollution Automobiles	

PRICES SUBJECT TO CHANGE

18. DISTRIBUTION STATEMENT	19. SECURITY CLASS (This Report)	21. NO. OF PAGES
Unlimited	Unclassified	110
	20. SECURITY CLASS (This page)	22. PRICE
	Unclassified	$5.50

MITRE Technical Report
MTR-7188

Air Pollution Assessment of Benzene

P. WALKER

APRIL 1976

CONTRACT SPONSOR	Environmental Protection Agency
CONTRACT NO.	68-02-1495
PROJECT NO.	077B
DEPT.	W-54

THE MITRE
McLEAN, VIRGINIA 22101

j(a)

Department Approval: _____

MITRE Project Approval: _____

ABSTRACT

Benzene acts primarily as a central nervous system depressant although it has also been shown to cause hematopoietic dysfunction and myelotoxicity in humans and animals. Production of benzene in the U.S. can utilize both coal and petroleum; however, the majority of the production is petroleum derived. Exposure of the human population to benzene can ensue not only from production facility emissions, but from usage of the compound, from oil spills, gasoline evaporation, and coke oven emissions. Control technology has been developed which, if utilized, can provide protection to industrial workers.

ACKNOWLEDGMENT

The author wishes to acknowledge those who gave generously of their assistance and time during the compilation of this document (one month time period). They include Mike Jones, Richard Johnson, and Justice Manning of the U.S. Environmental Protection Agency, and Linda Duncan, Edward Preston, Dr. Robert Ouellette, Dr. Lydia Thomas, and Dr. Bernard Baratz of The MITRE Corporation.

TABLE OF CONTENTS

		Page
I.	SUMMARY, CONCLUSIONS, AND RECOMMENDATIONS	1
II.	AIR POLLUTION ASSESSMENT REPORT	10
	A. PHYSICAL AND CHEMICAL PROPERTIES	10
	B. EFFECTS	21
	1. Humans	21
	2. Animals	27
	3. Other Effects	39
	C. AMBIENT DATA, POPULATION AT RISK AND MEASUREMENT TECHNOLOGY	45
	D. SOURCES	60
	E. CONTROL	84
REFERENCES		89

LIST OF FIGURES

Figure Number		Page
1	STRUCTURAL REPRESENTATIONS OF BENZENE	13
2	SUBSTITUENT PLACEMENT FOR BENZENE	17
3	COMMERCIALLY IMPORTANT REACTIONS OF BENZENE	18
4	GEOGRAPHIC DISTRIBUTION AND RELATIVE CAPACITIES OF BENZENE PRODUCTION FACILITIES	64
5	DISTRIBUTION AND RELATIVE CAPACITIES OF BENZENE PRODUCTION FACILITIES ON THE TEXAS AND LOUISIANA COASTS	65
6	COMMERCIAL USES OF BENZENE AND BENZENE DERIVATIVES	69
7	THE FLOW OF BENZENE THROUGH THE UNITED STATES IN 1971	71

TABLE OF CONTENTS (CONCLUDED)

LIST OF TABLES

Table Number		Page
I	PHYSICAL AND CHEMICAL PROPERTIES OF BENZENE	11
II	SPECIFICATIONS FOR COMMERCIAL BENZENE	15
III	REPORTED EFFECTS OF BENZENE EXPOSURE ON HUMANS AND ANIMALS	35
IV	REPORTED ACUTE BENZENE TOXICITY TO AQUATIC ANIMALS	40
V	REPORTED SUBLETHAL BENZENE TOXICITY TO AQUATIC ANIMALS	42
VI	MONITORING DATA FOR BENZENE	46
VII	SUMMATION OF TECHNIQUE FOR ANALYSIS OF BENZENE IN AIR AND WATER SAMPLES	55
VIII	U.S. BENZENE PRODUCTION CAPACITY (1975)	61
IX	CONSUMPTION OF BENZENE	72
X	UPPER LIMIT OF THE AMOUNT OF BENZENE LOST FROM BYPRODUCT MANUFACTURING FACILITIES IN 1971	74
XI	LOCATION OF PLANTS UTILIZING BENZENE AS AN INTERMEDIATE IN THE PRODUCTION OF SEVEN CHEMICALS	75
XII	BENZENE EMISSIONS TO THE ENVIRONMENT--1971 (IN MILLIONS OF POUNDS)	82

AIR POLLUTION ASSESSMENT OF BENZENE

I. SUMMARY AND CONCLUSIONS

A. PHYSICAL AND CHEMICAL PROPERTIES

Benzene is a very flammable, volatile, nonpolar, nonphotoreactive, cyclic aromatic hydrocarbon, often used as solvent for organic compounds. Under standard conditions, it is a clear, colorless, noncorrosive liquid, having a strong odor similar to that of gasoline. It maintains relatively high solubility with water, and is miscible with acetone, alcohol, chloroform, and carbon tetrachloride.

Benzene stability is enhanced by the alternating unsaturated bonds that create the characteristic resonating ring. In chemical reactions, benzene behaves primarily as a nucleophilic agent, usually undergoing substitutive, rather than additive, reactions. Common addition reactions include halogenation, hydrogenation, and oxidation. Sulfonation and nitration constitute the primary substitution reactions.

Because of the symmetry of the benzene molecule, only one monosubstitution product is possible. A large number of di-substitution products are known, but only three molecular arrangements are possible. Substitutions may be either adjacent (ortho), separated by one CH position (meta), or at opposite corners of the molecule (para).

B. EFFECTS

Benzene is rapidly absorbed by the blood through the lungs following inhalation with 28 to 50 percent of the inhaled vapor retained by the blood, with subsequent tissue fixation. Of this absorbed material, approximately 50 percent is eliminated unchanged via the lungs, with very small amounts (0.1 to 0.2 percent) excreted unaltered via the kidneys. The remaining benzene is oxidized to phenols and excreted as such or conjugated with sulfate ions in the liver and excreted as esters of glucoronic and sulfuric acids.

Inhalation of benzene vapor by humans has evoked central nervous system disorders, hematopoietic dysfunction, and death. A single exposure of 5 to 10 minutes in an atmosphere of 20,625 ppm has been shown to be fatal. Benzene poisoning at this concentration is characterized by its anaesthetic effect, due to the high affinity of brain lipids for benzene, which progresses from intoxication with possible convulsions or paralysis, to loss of consciousness followed by coma and death. Postmortem analysis has revealed extensive hemorrhaging, respiratory tract infections, kidney congestion, hyperplasia* and hypoplasia** of sternal bone marrow, and cerebral edema. Similar effects have been reported for other mammalian species.

At nonlethal concentrations, the human central nervous system is disrupted to varying degrees, depending upon extent of exposure.

---

*Abnormal increase in cell number.

**Defective development.

Some of the physical symptoms include insomnia, agitation, headache, dizziness, drowsiness, breathlessness, irritability, vertigo, and nausea. Brain electropotential has been shown to increase at an atmospheric concentration of 0.875 ppm benzene, which is the odor threshold. This may indicate the inefficiency of olfactory detection of danger; however, the medical significance of this effect has not been determined. Upon exposure to benzene vapor, nonhuman mammalian subjects have exhibited depressed reflex strength, increased latent period to a positive stimulus, decreased incidence of spontaneous behavior, and changed conditioned reflex patterns.

The most unique clinical aspect of chronic benzene poisoning is hematopoietic system damage. Anemia, characterized by decreased red blood cell count; thrombocytopenia, a reduction in platelet numbers, leukopenia, a depression of white blood cell count; and low hemoglobin concentrations are often exhibited in patients suffering benzene poisoning. Inhalation of 88 ppm, for 204 days, caused histopathological alterations in rats, with a similar effect on guinea pigs after 269 days. Rabbits exhibited these same symptoms after 243 days' exposure to 80 ppm. Slight leukopenia was reported in rats exposed to 44 ppm for 20 hours per week for 5 to 7 weeks. Qualitative alteration of the hematopoietic system has included cholinesterase activity reduction, leukocytosis, lower leukocyte DNA values, stimulated bone marrow erythrocyte activity, and reversal of polymorphonuclear/lymphocyte ratio. Lower hemoglobin concentrations and minor hematological deviations

have been reported in workers exposed to maximum atmospheric concentrations of 25 ppm, perhaps indicating a hematopoietic effect threshold.

Threshold doses have been shown to alter blood chemistry and composition in rats. Increases in cytochrome P450 and aminopyrine demethylase activity were reported in rats after ten 5-hour per day inhalation exposures to 450 to 500 ppm. It was concluded that prolonged exposure, even at low concentrations, may alter the biotransformation of foreign substances and endogenous compounds by the liver. Because of the detoxification role played by the liver, this alteration could have serious ramifications for the affected organism.

Chronic benzene poisoning, manifested by leukopenia, has been shown to lower the resistance of rabbits to pneumonia and tuberculosis. The phagocytic index and phagocytic number were found to decrease in rabbits at an atmospheric level of 0.02 mg/l (6.25 ppm), which is less than the allowable limit of 32 mg/m^3 (10 ppm) established by NIOSH. The normalization of phagocytic activity did not occur until three months after exposure to 0.05 mg/l (15.625 ppm).

Significant chromosome aberrations have resulted from chronic benzene inhalation in both humans and other mammals. Peripheral blood lymphocyte studies of 25 human patients who recovered from benzene hemopathy revealed increased stable as well as unstable chromosome alterations. The stable deviations were still present several years

after cessation of exposure, with a decrease in the number of unstable changes persisting.

The stable bone marrow chromosome aberrations may be indicative of leukemic clone induction since chromosome alterations have been reported in patients with a history of benzene exposure followed by diagnosis of leukemia. One of the best studies in support of this possibility is a benzene-associated leukemia study from Japan, in which 413 leukemia patients were matched with a control case based on particular parameter.. Ten of the patients in the study worked at occupations involving benzene exposure and these collective patients were associated with an increased leukemic risk. Carcinogenic studies have also been conducted on animals, but no conclusive evidence has been found due to lack of proper experimental controls, statistically nonsignificant data, or parallel occurrence of leukemia in control groups.

C. AMBIENT DATA

In response to complaints during 1974, the Occupational Safety and Health Administration analyzed 269 industrial atmospheric samples for the presence of benzene. However, a summary of the test results from those samples is not readily available. The expected concentration of benzene in urban air is reported to be 0.01 to 0.05 ppm, although the hydrocarbon is not routinely monitored in the atmosphere. Of the 12 reported benzene detections recorded in this report, the concentration range was from a "trace" to 0.098 ppm, with one excursion to 23 ppm reported in the vicinity of a solvent

reclamation plant. Although a trace of benzene was found in finished waters from the Mississippi River, it was not detected in the effluent from any of the facilities on the river, suggesting a contamination source other than industrial waste.

D. POPULATION AT RISK

To assess the risk of atmospheric benzene presented to the general population, a hypothetical production facility, with a yield of 185 million gallons of benzene annually, would theoretically lose 0.37 million gallons of this benzene to the atmosphere in the form of flue gases. This would result in an ambient benzene concentration 500 meters downwind of the plant of 2.7 mg/m^3, or 0.845 ppm.

In determination of the effect of this atmospheric concentration of benzene on humans, 2.8 mg/m^3, or 0.875 ppm, of benzene has enhanced brain electropotential; however, the significance of this alteration of cerebral activity has not been established. NIOSH has established a maximum time-weighted average exposure of 10 ppm for the worker exposed to benzene. In order to equal this maximum recommended level, an individual would have to remain downwind of the hypothetical production facility for 20 days. Therefore, if the NIOSH exposure level precludes deleterious effect, the facility would not be emitting a significantly hazardous concentration of benzene.

E. MEASUREMENT TECHNOLOGY

Various acceptable and adequate methodologies for collection and detection of ambient benzene have been developed. Silica gel with a prefilter of molecular sieves, activated charcoal, grab samples, bubblers, and the use of nitrating solutions for absorption in scrubbers have all been utilized. Many techniques for extraction and/or preconcentration of benzene samples exist with alterations reported by virtually all investigators. Analytical detection methods include direct ultraviolet spectrophotometry, colorimetry, and gas chromatography, which is the most accurate and sensitive.

F. SOURCES

Benzene is produced commercially in the United States from both coal and petroleum, but since 1959 petroleum has been the major source. The total domestic benzene production capacity for 1975 is 1,743 million gallons (1.7×10^9) annually. Crude benzene is also imported into the United States at a rate of approximately $110,000 worth in 1974, while an average of 563×10^6 pounds of chemical benzene are imported each year.

Benzene can be commercially synthesized by several methods. In the coal coking process, coal tars may be distilled to yield various products, including a benzene fraction. It can also be isolated from the light oil that is recovered from coking operations. However, catalytic reforming of petroleum feedstocks produces approximately 70 percent of the benzene obtained from petroleum refineries.

Prior to World War II, the major use of benzene was in gasoline blends, but presently it is consumed primarily in non-fuel uses, usually as a raw material for by-product synthesis (i.e., ethylbenzene, phenol, cyclohexane). These intermediate substituted benzene chemicals represent the major consumption of benzene and are potential sources of benzene emission into the atmosphere.

Two additional sources of potential benzene contamination not arising from either commercial production or use are oil spills and motor vehicle emissions. It is calculated that 22 to 24 million pounds of benzene are released annually into oceans and waterways from spills, while gasoline evaporation and consumption release approximately one billion pounds to the environment. Another source of environmental benzene is coke ovens. A calculated 122 million pounds of benzene were released to the environment based upon 1974 estimates of coke production. Environmental transport of benzene from the point of emission has not been clearly defined. Due to its high solubility in water, atmospheric benzene could be introduced to the water via rainfall, after which volatilization to the atmosphere could occur. Therefore, a continuous cycling between the air and water is likely to occur, indicating significant environmental mobility and possible dispersion and distribution ability. Due to the long-term stability of benzene, a cumulative effect may take place.

G. CONTROL

Adequate, efficient, and simple measurement technology has been developed that, if employed, can provide protection to industrial workers. Benzene emissions can be controlled by using a vapor condensation apparatus in the form of scrubbers and separators in the catalytic reforming process. Routine maintenance of these mechanical additions would help eliminate the risk of high ambient benzene concentrations. Proper ventilation of the areas in a facility that utilizes benzene and isolation of these areas from other work regions would further reduce the possibility of hazardous benzene concentrations accumulating. Proper handling and transport techniques, if emphasized, add to the safety of not only the industrial worker, but also the general population.

Additional benzene emissions may be reduced at industrial sites by substituting process materials and/or equipment. If this approach were operationally and economically acceptable, it could effectively achieve zero benzene emissions. However, careful consideration must be given to the health hazard that may result from the new material or equipment.

The problem posed by oil spills and automotive exhaust is more ubiquitous and would probably be affected only by legislation imposing stiff penalties and/or fines on the involved companies (i.e., oil producers and transporters, vehicle manufacturers, and gasoline manufacturers).

II. AIR POLLUTION ASSESSMENT REPORT

A. PHYSICAL AND CHEMICAL PROPERTIES

Benzene is a nonreactive, nonpolar, highly refractive cyclic aromatic hydrocarbon. Under standard conditions, benzene is a clear, colorless, noncorrosive, nonphotoreactive, highly flammable liquid. It has a strong odor similar to that of gasoline. It is often employed as a solvent for organic compounds and as an ingredient of motor fuels. It is relatively soluble in water and is miscible with acetone, alcohol, chloroform, ether, carbon disulphide, carbon tetrachloride, glacial acetic acid, and oils. Reactivity does occur with bases, but only slowly with oxidizing agents. Pertinent physical properties of benzene are listed in Table I (1, 2, 3, 4).

Benzene stability is a result of the alternating unsaturated bonds that are capable of resonating position. X-ray and electron diffraction studies have indicated that the six carbon atoms comprising benzene are arranged symmetrically in a plane forming a regular hexagon. The hydrogen atom attached to each carbon atom in turn forms a doughnut-shaped electron orbital above or below the plane (termed a super Pi bond)(4,5).

Several structural representations have been proposed to pictorially illustrate this arrangement of the compound, but the most realistic and commonly used is a hexagon with a circle in the center (see Figure 1 C). Kekulé ring formulas are also provided in Figure 1. Both complete and abbreviated styles occasionally used,

TABLE I

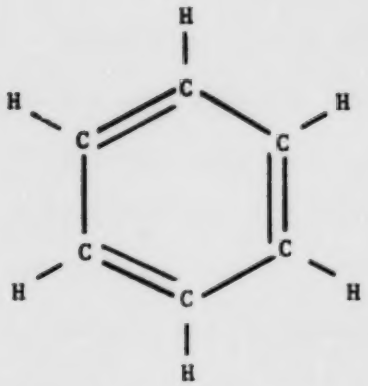
PHYSICAL AND CHEMICAL PROPERTIES OF BENZENE

Molecular formula	C_6H_6
Physical state	liquid
Molecular weight	78.11
Melting point	5.533°C
Boiling point	80.103°C
Specific gravity	0.8787 15°/4°C
Refractive index	1.5016 at 29°C
Vapor pressure	100 mm Hg at 26°C
Partition coefficient (octanol/water)	2.14 average
Weight/gallon	7.32 lbs.
Flash point, closed cup	12°F
Autoignition temperature	1044°F
Flammable limits in air (percent by volume)	1.35-7.90
Vapor density (air = 1)	2.7
Percent in saturated air (760 mm)	13.15 (26°C)
Density of saturated vapor--air mixture at 760 mm (air = 1)	1.22 (26°C)
Specific dispersion	189.6
Conversions	1 ppm = 3.19 mg/m^3 25°C 1 mg/liter = 313 ppm
Odor	100 ppm = intensity of 1-2

TABLE I (CONCLUDED)

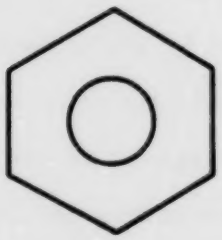
Viscosity (absolute)	0.6468 at 20°C, cp
Surface tension	28.18 at 25°C, dyne/cm
Critical temperature	289.45°C
Critical pressure	48.6 atm
Critical density	0.300 g/ml
Heat of fusion	2.351 kcal/mole
Heat of vaporization	8.090 kcal/mole at 80.100°C
Heat of combustion (liquid C_6H_6 to liquid H_2O and gaseous CO_2)	9.999 kcal/g at constant pressure and 25°C
Solubility in water	0.082 g/100 ml at 20°C
Solubility of water in benzene	0.05 g/100 g benzene at 25°C

FIGURE 1
STRUCTURAL REPRESENTATIONS OF BENZENE



A. Kekule Ring Formula

B. Abbreviated Kekule Ring Formula



C. Standard Formula

as well as the hexagonal depiction commonly used are presented. The hexagonal structure implies the presence of one carbon atom at each of the six angles and a hydrogen atom at each carbon. The C-C bond lengths are 1.35 Å, while C-H bond lengths measure 1.08 Å (4, 5).

Water solubility and vapor pressure are significant parameters in environmental occurrence. Values of these parameters for benzene at 26.085°C are 1800 ppm and 100 mm Hg, respectively. While reviewing reported solubilities of benzene in water, a variation of 1730 to 1800 ppm was noted and attributed to either temperature variances or differences in technique precision (6). Salting-in and salting-out effects for benzene in aqueous solution were reported by Giacomelli and Spinetti (7). Comparison of saltwater and distilled water solubilities of benzene reveals a decrease in ppm dissolved as the salt content of the water increases (8, 9). These data indicate that benzene could be environmentally distributed in the aqueous phase as well as in the gaseous phase.

Commercially available benzene with a boiling range equal to or less than 1°C has been analyzed by gas chromatography and shown to contain no more than 0.2 percent, and often no more than 0.1 percent, hydrocarbon impurities. These impurities include olefinic, paraffinic, and cycloparaffinic hydrocarbons and toluene, as well as traces of tiophene and carbon disulfide (4). Specifications for three commercial grades of benzene can be found in Table II (10, 11).

TABLE II

SPECIFICATIONS FOR COMMERCIAL BENZENE

	Industrial Grade	Refined	Refined-Nitration Grade
Distillation range at 760 mm	no more than 2°C, including 80.1°C	No more than 1°C, including 80.1°c	No more than 1°C, including 80.1°C
Specific gravity 15.56/15/56°	0.875-0.886	0.8820-0.8860	0.8820-0.8860
Thiophene as a contaminant	--	1ppm	--
Nonaromatics as contaminants	--	0.15% maximum	--

In chemical reactions, benzene behaves primarily as a nucleophilic agent, usually undergoing substitution of individual hydrogen atoms rather than addition. The two most common substitutive reactions are nitration and sulfonation. Additive reactions are of three types: oxidation, which only occurs under extreme conditions; hydrogenation; and halogenation, which like nitration, is irreversible. Because of the aforementioned symmetry of the benzene molecule, only one monosubstitution product is possible. A large number of disubstitution products are known. For any two substituents, three arrangements are possible (see Figure 2). Groups may be adjacent to each other (ortho), separated by a single CH (meta), or at opposite corners of the benzene ring (para) (4, 5).

The position of substituents in tri- or polysubstituted benzenes is usually indicated by numbers. As an example, the trichlorobenzene in which all three chlorines are adjacent is called 1,2,3,-trichlorobenzene (3, 5, 12).

Chemical reactivity for benzene is highly limited due to the aforementioned stability of the ring structure. Therefore, commercial chemical reactions with benzene normally require catalysts, strong acids, elevated temperature, or elevated pressure. These are illustrated in Figure 3 (4). Benzene is an important intermediate in the production of: ethylbenzene (raw material for styrene), nitrobenzene (raw material for aniline); cyclohexane; chlorobenzene (raw material for DDT); dichlorobenzenes; and maleic anhydride (4).

FIGURE 2
SUBSTITUENT PLACEMENT FOR BENZENE

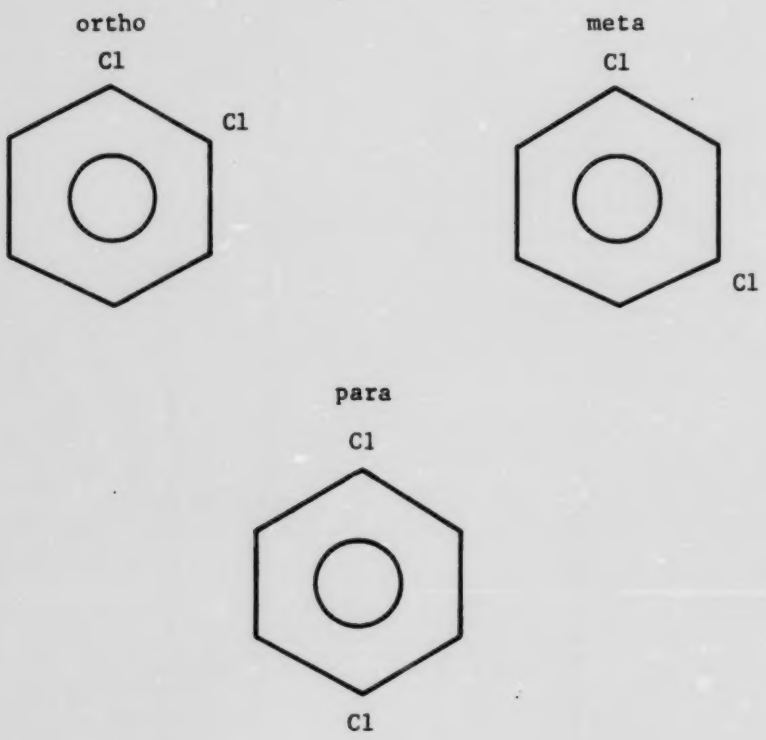
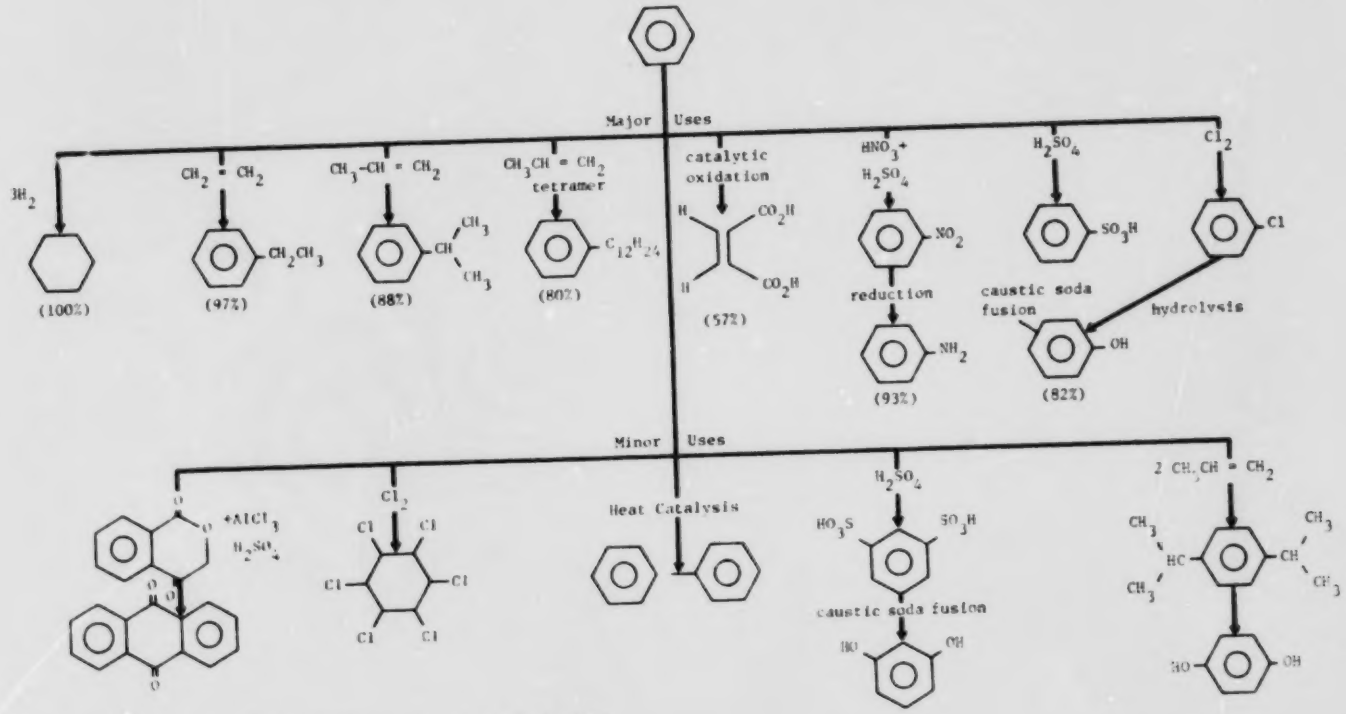


FIGURE 3
COMMERCIALLY IMPORTANT REACTIONS OF BENZENE*



Source: Erskine, M. G., Chemical Economics Handbook, 1972.

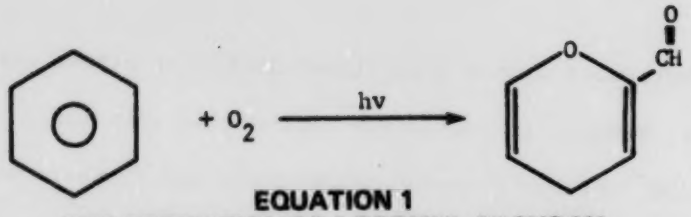
*Numbers in parentheses indicate yields based on benzene starting material.

Unless substituted with a significant number of strong electronegative groups, benzene is completely nonreactive with water or hydroxyl ions (OH^-) unless elevated temperature and pressure are conditions of the reaction. Commercial hydrolysis of monosubstituted benzene species (e.g., chlorobenzene) is much more easily effected than direct hydrolysis of normal benzene.

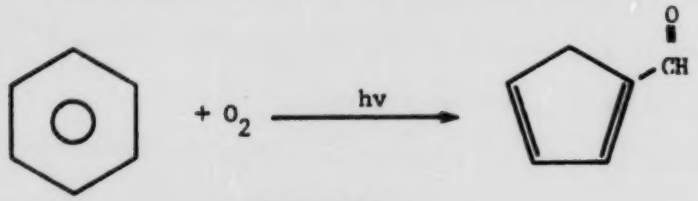
Oxidation of benzene to a number of different compounds can occur, but catalysts and/or elevated temperature and pressure are normally necessary. As an example, permanganate or chromic acid, both oxidation agents, react with benzene to form water and carbon dioxide only under rigorous conditions.

Benzene dissolved in cyclohexane does not directly absorb appreciable amounts of light at wavelengths longer than 280 nm (2800 Å), while gaseous benzene does not photoreact to wavelengths greater than 275 nm. Since the atmospheric ozone layer effectively filters out light wavelengths less than 290 nm, it appears unlikely that excitation and photoreaction of environmental benzene will occur. However, indirect reaction may be possible due to the presence of sensitizers when the benzene is in water or soil (13).

Photolysis of an oxygenated aqueous solution of benzene has been reported using <290 nm light. The formation of 2-formyl-4H-pyran (as in equation 1) was reported using 253.7 nm light, but a later study identified the photoproduct as cyclopentadiene-carboxaldehyde (see equation 2) (14, 15). The formation of acetylene, ethylene, methylacetylene was recorded using 147 nm wavelength on benzene vapor that



EQUATION 1
THE FORMATION OF 2-FORMYL-4H-PYRAN



EQUATION 2
THE FORMATION OF CYCLOPENTADIENE-CARBOXALDEHYDE

had been degassed under vacuum. Although the previously mentioned compounds were produced, the major product was a polymeric material (16). Benzene vapor photolyzed in the presence of oxygen has produced different compounds, depending on the solvent used as a trapping agent. Organic solvents and water were experimentally utilized in the trap with respective detection of mucodialdehyde and phenol (17). Atomic oxygen, generated from the photodecomposition of the nitrogen dioxide frequently found in high concentrations in heavily polluted areas, has been shown to react with benzene to form phenol (18, 19).

The detection of benzene in heavily polluted air has led to the experimental photolysis of the hydrocarbon under simulated smog conditions. Although considerable variations in reaction conditions have been utilized, benzene has been characterized as one of the least reactive hydrocarbons in terms of hydrocarbon loss (18). Some slow reaction has been reported using short-term studies; however, the long-term stability of benzene is virtually established.

B. EFFECTS

1. Humans

Benzene is readily absorbed by the blood through the lungs, with 28 to 50 percent of the inhaled vapor retained by the blood. Fifty-five to 60 percent of this absorbed material becomes fixed in marrow, liver, and fatty tissues and remainder is exhaled unchanged (20, 21, 22). Saturation of the circulating blood occurs rapidly, 70 to 80 percent in 30 minutes, with tissue saturation levels not reached for several days (23).

Approximately 50 percent of the absorbed benzene is eliminated unchanged via the lungs, with small quantities, 0.1 to 0.2 percent, excreted by the kidneys in an unaltered state (20, 22). The concentration of benzene in the exhaled air decreases rapidly with time after cessation of exposure (24). The remaining benzene is oxidized to phenols and diphenols and excreted as such or conjugated with sulfate ions in the liver and excreted as esters of glucuronic and sulfuric acid within 48 hours (21, 22).

Ingestion, dermal contact, and inhalation are the three modes of benzene exposure expected to affect humans. Oral applications due to accidental ingestion can lead to illness or death (25, 26). Mucous membrane irritation develops upon exposure, and absorption of the benzene into the blood is characterized by symptoms of systemic intoxication (27). Dermal contact has not been reported to be a serious hazard, although benzene can be slightly absorbed through

the skin. Lack of observable urinary sulfate changes and absence of benzene in exhaled air from three men dermally exposed to benzene for 25 to 35 minutes support the assumption that systemic poisoning from brief skin exposure is not probable (28). However, caution should be applied because erythema,* skin defatting, dry scaling, and secondary infections have been noted after dermal benzene contact (23).

Inhalation of vapor is the most common form of exposure, due to the presence and volatility of benzene in motor fuel, paint, rubber cement, adhesives, and dry cleaning fluids and its uses in the chemical, coke, printing, and lithography industries (23). A single exposure of 5 to 10 minutes in an atmosphere of 66 mg/l (20,625 ppm) has been fatal. This fatality due to benzene is characterized by its anesthetic effect, caused by the high benzene affinity for brain lipids, which progresses from intoxication, with possible convulsions or paralysis, to loss of consciousness followed by coma and death (29).

Many cases of fatal inhalation of benzene have been reported (30, 31, 32) and most are caused by vapor concentration increases in confined areas (33). Postmortem analysis has revealed extensive petechial** hemorrhage in tissues of the brain, pleurae, pericardium, urinary tract, mucous membranes, and skin (27, 34). Respiratory

---

*Diffused redness over the skin caused by capillary congestion.

**Small purplish spots.

tract infections, hypoplasia* and hyperplasia** in sternal bone marrow, kidney congestion, and cerebral edema*** organs and observed (35, 36).

At nonlethal concentrations, the hematopoietic*** organs and the central nervous system are affected, with variations due to exposure time and concentrations. Some physical symptoms associated with this lower level exposure include insomnia, agitation, headache, heaviness of head, dizziness, drowsiness, breathlessness, unsteadiness, irritability, vertigo, nausea, and loss of appetite (23, 26, 37). Exposure to benzene at a concentration equal to the odor threshold, 2.8 mg/m^3 (0.87 ppm), has been shown to enhance brain electropotential. This fluctuation in brain electropotential, which is of undetermined significance, was reported to cease at a concentration of 1.5 mg/m^3 (0.46 ppm) (38). However, clinically detected benzene poisoning has been reported with none of these accompanying symptomatic indications (39).

Chronic benzene poisoning, resulting from prolonged exposures to greatly fluctuating ambient concentrations ranging from 25 ppm to an estimated 5320 ppm, has been frequently reported and studied. Susceptibility has not been found to be either age- or sex-related (40, 41, 42). The most unique clinical aspect of this condition is

---

*Defective tissue development.

**Excessive tissue formation.

***Condition in which the tissue contains an excessive amount of fluid.

****Blood-making.

the injury to the hematopoietic system. Anemia, characterized by decreased red blood cell count; thrombocytopenia, a reduction in platelet numbers; leukopenia, a depression of white blood cell count and low hemoglobin concentrations are often exhibited in patients suffering benzene poisoning. Occasionally, increased cell size or an elevation of eosinophil* counts occur (35, 36, 42). A study of the blood coagulation system during chronic benzene poisoning has shown that hemorrhaging is caused primarily by the thrombocytopenia coupled with thrombocytopathy, a qualitative deficiency of platelets, which lowers system activity level, increases fibrinolysis,** disturbs endothelial barriers, and increases the permeability of the vascular walls (43). The abovementioned alterations of the hematopoietic system may account, in part, for the increased susceptibility to nonoccupational diseases found in workers with benzene poisoning (44).

Another important finding correlated with benzene poisoning from unspecified ambient concentrations has been an increase in chromosome aberrations, both in vitro, as lymphocyte cell cultures, and in vivo (45). Studies of peripheral blood lymphocytes in 25 subjects who had recovered from benzene hemopathy,*** four subjects

---

*Cells, part of the white blood cell complement that originate in the bone marrow and are readily stained with eosin, an acid stain.

**Conversion of fibrin into soluble substances by proteolytic enzyme action.

***Blood disease.

with bone marrow toxicity, and three who had recovered from acute benzene poisoning determined significant increases of stable and unstable chromosome aberrations (45).

Cytological examinations of workers exposed to benzene in a rotogravure plant correspond to these (46), as did a report on workers in a factory employing benzene as a solvent (47). These chromosomal deviations were still present several years after exposure cessation and recovery; however, a decrease in the number of unstable changes and a persistence of the stable ones were observed, with an occasional abnormal clone found in peripheral blood lymphocytes (45). Another pertinent study of chromosomal aberration was conducted on a pregnant woman, suffering benzene-induced aplastic anemia* with severe hemorrhagic side effects, who delivered a normal male. Cytogenetic evaluations of the mother showed an increased rate of chromosome aberrations, while the infant was normal (45).

The persistent chromosome changes seem simply to indicate damage occurrence. However, stable bone marrow chromosome aberrations have been reported in patients with a history of benzene exposure followed by development of leukemia (48, 49, 50). However, these implications have not yet been resolved with respect to the problem of benzene-associated leukemia (43, 45).

---

*A reduction in the number of circulating red blood cells or hemoglobin, or both, caused by failure of the bone marrow to function.

Benzene of undetermined or fluctuating concentration has been suspect in cases of leukemia since the 1930's (51, 52). One of these associations was not supported by early poisoning symptoms or hematological studies due to patient fatalities. Of the 19 cases reported in a second study, two were verified leukemics, one with the acute myeloid type and one with leukemic marrow replacement. In another benzene-associated leukemia implication, there were not sufficient specific environmental data to confirm the hypothesis (53). In 1939, one case of acute myeloblastic leukemia and one of lymphoblastic leukemia were reported in patients with prolonged benzene exposure (54). In the 1960's, the association of benzene exposure and development of leukemia after a latent period was reported by many researchers. DeGowin (55) outlined the history of a patient having aplastic anemia, which was benzene associated, who developed acute myeloid leukemia 15 years later. Tareeff noted a latent period in three cases of acute leukemia. Many more reports of benzene exposure followed by a diagnosis of leukemia at a later time surfaced in the 1960's and early 1970's (56, 57, 58).

One of the most significant, and perhaps best designed, studies presented is a case-control benzene-leukemia study from Japan. All diagnosed leukemia patients from 1945 to 1967, living in Hiroshima or Nagasaki City at the time of disease diagnosis, were included. Four hundred and thirteen of these cases were matched with control cases based on four parameters: sex; date of birth ±30 months;

distance from the atomic bomb explosion; and living in either city, at the time of the patient's diagnosis. Within this evaluation, ten occupations involved benzene exposure, and these collective patients were associated with an increased risk for leukemia. Twenty-four of these cases were too far from the A-bomb explosion to attribute the disease to radiation; however, all of the occupations could have involved exposures other than benzene (59).

It is difficult to correlate medical findings in exposed humans with airborne benzene concentrations due to lack of adequate environmental data during exposure. Some reports contain estimated or possible levels, but even these are sketchy. Flury did delineate levels for a single exposure with the risk involved: (29)

3,000 ppm—endurable for 30-60 minutes

7,500 ppm—dangerous after 30-60 minutes

20,000 ppm--fatal after 5-10 minutes

Two other studies facilitate determination of degree of danger. Greenberg reported chronic benzene poisoning, evidenced by leukopenia, in patients exposed during work hours to 100 ppm (60). Lower hemoglobin concentrations and minor hematological deviations were discovered in workers exposed to maximum concentrations of 25 ppm, perhaps indicating a hematopoietic effect threshold (61).

2. Animals

The effects of benzene on laboratory animals have closely paralleled many of the manifestations seen in man. Schrenk et al. found a linear relationship between benzene concentrations

in the air and the equilibrium concentration in the blood of dogs (62). As in humans, the initial blood absorption was rapid, almost complete within 30 minutes, with subsequent tissue absorption and distribution occurring for several hours. Plasma manifested the lowest benzene concentrations, with progressive increases in concentration levels from red blood cells to muscle and organ tissues. Fatty tissue, bone marrow, and urine were found to contain the highest levels, 20 times the concentration found in blood. Benzene has been shown to be extremely soluble in the fatty tissue of rabbits, 30 to 50 times as in blood, with tissue-blood partition coefficients for most body tissues in the range of 1 to 3 (63). A similar solubility was reported for rats, with distribution of benzene occurring in central nervous system tissues, bone marrow, and liver (64).

Phenol has been found to be the primary metabolite of benzene in animals, with hydroquinone and catechol in lesser amounts. Phenol is usually conjugated with glucuronic acid and sulfates in the blood and excreted in this form by mammals. Often the toxic effects of benzene have been attributed to these metabolites (65, 66, 67). However, subcutaneous injections of these materials, even at doses approaching LD_{50}'s,* have failed to produce any hematopoietic toxicity, which is a primary effect of benzene exposure (68).

During inhalation experiments, a latency period has been demonstrated prior to the development of toxic effects. This latency

*A dose lethal to 50 percent of the exposed population.

period was found to increase in rats with a graded reduction of benzene concentration (from 5000-50 mg/m^3 or 1562.5 - 15.625 ppm). The latency period is also lengthened by increased time intervals between exposures (69). During acute inhalation experiments, 3 of 8 male Long Evans rats died 24 hours after 5 exposures to 40,000 ppm, each exposure lasting 20 to 35 minutes. Death was reported in 2 of 10 rats exposed to 10,000 ppm for 12.5 to 30 minutes daily for 1 to 17 days (70). A no-effect level for rats, guinea pigs, and rabbits was found to be below 88 ppm after exposure for 7 hours/day for 269 days (71). Rats exposed to 44 ppm for 5 hours/day, 4 days/week, for 5 to 7 weeks have exhibited slight leukopenia (72).

The effects of exposure are primarily disruption of the hematopoietic and central nervous systems. Dogs have exhibited initial hypertension, followed by vasomotor paralysis due to the anesthetic effect of benzene inhalation on the smooth muscles of blood vessels. Irritation of mucous membranes with severe pulmonary* edema and hemorrhage was observed upon exposure to liquid benzene (73). Rats exposed to levels of 44 ppm, for 7 hours/day, 5 days/week, have shown significant leukopenia (72); with exposure of 200 ppm, 8 hours/day, 5 days/week, leukopenia was noted after 90 days (74). After 105 hours of exposure to 1000 ppm for 23.5 hours/day, 7 days/week, loss of body weight; nose and mouth hemorrhage; stomach distention; empty gut; and engorged blood vessels in the lung, liver, kidney, intestine,

*Related to the lung.

and omentum were observed. Two-hundred and four days' exposure to 88 ppm caused histopathological alterations in blood, bone marrow, spleen, and testes. Guinea pigs exhibited the same histopathological alterations after 269 days. Leukopenia and degeneration of seminiferous tubules* affected rabbits after 243 days' dosage of 80 ppm (73). Rabbits exposed to 35,000 to 45,000 ppm were lightly anesthetized after 3.7 minutes, experienced excitation and tremors after 5 minutes and died after 36 minutes (75).

Qualitative, as well as quantitative alteration of the hematopoietic system has been observed. Reduction of cholinesterase activity, leukopenia,** leukocytosis,*** lower leukocyte DNA values, and stimulated bone marrow erythrocyte activity have been reported infrequently (59, 69, 74, 76, 77, 78). Reversal of the polymorphonuclear:lymphocyte ratio occurred in rats with a pre-exposure ratio of 22:57 and a post-exposure ratio of 54:46 tabulated after a 105-day exposure to 1000 ppm, 23.5 hours/day, 7 days/week (74). Even threshold doses have altered blood chemistry and composition in rats (79).

In order to clarify the state of hematopoietic depression in bone marrow associated with benzene poisoning, the metabolic features of the nucleic acids, which relate closely to cell proliferation,

---

*Passage containing sperm found in the testes.

**Reduction in the number of white blood cells in the blood.

***Increase in the number of white blood cells in the blood.

were studied in rabbits with benzene poisoning. Bone marrow tissue was found to turn fatty and showed a significant decrease in cell number. The amounts of nucleic acid, particularly DNA, per unit weight of tissue decreased. The ratios of cytoplasmic RNA and ribosomal RNA to DNA both increased approximately twofold. The synthesis rates of both RNA's and DNA also increased (80).

Because of the hematopoietic effects reported and due to the function of the central nervous system as a regulation of hematopoietic activity, effects of benzene exposure on the central nervous system have been delineated. A delay in conditional response time was reported in rats exposed to 20 ppm for 6 hours/day, 6 days/week, for 5 1/2 months (81), but not in rats exposed to 4 ppm for the same time period. Mice exposed 6 hours/day for 20 days to benzene concentrations of 100 ppm showed a decrease on the fifth day in wheel-turning activity, which is normally spontaneous behavior. It was concluded from observations made during this experiment that the no effect level for mice was below 10 ppm (82). Changed conditioned reflex patterns as well as depressed reflex strength have also been observed in laboratory animals after exposure to benzene (64, 77, 76, 83). Jonek, in an explanation of the effects of benzene on the central nervous system, suggested a selective influence of the hydrocarbon on neuron oxidation.* Due to enzymatic changes observed in mice

*Neurons transmit nerve impulses by transmitting electrons; interference with this process is theorized to result in anesthesia.

exposed to a one-time concentration of 18,750 ppm, Jonek indicated a possible direct lesion of the lipoprotein membranes, which would alter the neuron oxidation (84).

Benzene has been reported to have other effects on animals. Slight increases in cytochrome P450 and aminopyrine demethylase activity were noted in male Wistar rats exposed to a benzene concentration of 450 to 500 ppm, 5 hours/day, for 10 days. It was concluded that with prolonged exposure, even low concentrations may alter the biotransformation of foreign substances and endogenous compounds by the liver (84). Oxidation-reduction processes and the albumin production in the liver were disturbed in rats exposed to a threshold dose (85). Marked hypertrophy of smooth endoplasmic reticulum of the tubular type, disappearance of segmental distension of the ergastoplasm,* tendency to ribosome loss and swelling, and myelin degeneration of mytochondria were observed in rabbits with subacute benzene poisoning after exposure to 10 mg/l(3125 ppm), 6 hours/day, for 14 days (86). Maternal exposure in rats produced significant biochemical changes in both the pregnant female and the fetus at concentrations ranging from 1 to 63.5 mg/m^3 (0.3125 to 19.84 ppm). The only measured dosage difference was an increase in utilization of of ascorbic acid that was linear with dosage (87).

Benzene exposure has been shown to lower species resistance to infection. Rabbits with benzene-induced leukopenia exhibited

---

*Functional protoplasm.

reduced resistance to pneumonia and tuberculosis, while active acute infections were found in rabbits previously injected with benzene (88-91). In a study of chronic benzene poisoning in rabbits, both the phagocytic index* and the phagocytic number** were decreased. These changes began earlier than hematopoietic alterations and occurred even at a level of 0.02 mg/l (6.25 ppm), which is less than the allowable limit of 32 mg/m^3 (10 ppm). The restoration of phagocytic activity was not normal until three months after exposure to 0.05 mg/l (15.625 ppm) (92).

Chromosome aberrations have been induced in the bone marrow cells of rabbits subcutaneously exposed to benzene. Of 16 experimental animals evaluated, the karyotype was normal in all but one. Early in the experiment the percentage of mitosis with chromosomal aberrations was 5.9; after 18 weeks the average increased to 57.8 percent. Gaps and breaks were frequently observed, with tetraploid mitosis occasionally seen (93).

Possible induced susceptibilities to the effects of benzene have been examined. The condition of pregnancy was not found to enhance the hematotoxic effect of benzene in guinea pigs. However, inadequate dietary protein intake was found to increase the susceptibility of dogs and rats to benzene poisoning (94).

---

*Average number of bacteria ingested per leukocyte.

**Number of cells having the ability to ingest and destroy foreign substances.

Variances in oral LD_{50}'s of benzene have been reported. Male Sprague-Dawley rats were reported to have LD_{50}'s of 0.93 (0.71-1.23) g/kg bodyweight in one study (62), while another report established 3.4 g/kg bodyweight in young adults and 4.9 g/kg bodyweight in older animals as the LD_{50}'s (95). An oral LD_{50} of 5.6 g/kg bodyweight was reported for male Wistar rats (73). A difference in species response to long-term benzene poisoning has also been reported in mice. After exposure to 200 ppm for 20 days, only one strain had leukopenia, but it did not exhibit the hemoglobin concentration decrease observed in the other strain. It was recommended that the more sensitive strain be used for determination of LD_{50}, acute response time, and threshold levels in mice (96).

Carcinogenic studies have been conducted on laboratory animals, but no conclusive evidence was found in the literature. Many negative reports used dermal application as the mode of application, but not all the possible tumor sites have been examined (97-99). Subcutaneous or intramuscular injections have also been employed in establishing the incidence of leukemia in mice. Unfortunately, absence of controls, statistically non-significant data, or parallel occurrence of leukemia in controls render the data insufficient (100-103). Long-term carcinogenic studies using inhalation as the method of exposure were not found in the available literature. The reported effects of benzene on humans and animals are summarized in Table III.

TABLE III

REPORTED EFFECTS OF BENZENE EXPOSURE OF HUMANS AND ANIMALS

Organism	Mode of Exposure	Concentration of Exposure (ppm)	Duration of Exposure	Effect	Reference
Human	Oral	--	Acute	Death	25, 26
	Oral	--	Acute	Mucous membrane irritation and systemic intoxication	
	Dermal	Immersion of tissue	Acute	Erythema, skin defatting, dry scaling, secondary infections	27
	Inhalation	20,000	5-10 minutes	Convulsions, paralysis, coma, and death	23
	Inhalation	High	Acute	Petechial hemorrhage in body tissues, respiratory tract infections, hypoplasia and hyperplasia in sternal bone marrow, kidney congestion, and cerebral edema, death	27, 34-36
	Inhalation	Sublethal	Chronic	Insomnia, agitation, headache, dizziness, drowsiness, breathlessness, unsteadiness, irritability, vertigo, nausea, loss of appetite.	23, 27, 37
	Inhalation	2.8 mg/m^3 (0.875 ppm) (odor threshold)	Acute	Brain electropotential enhancement	39
	Inhalation	Sublethal	Chronic	Anemia, thrombocytopenia, thrombocytopathy, leukopenia, low hemoglobin concentration, increased cell size, eosinophil count elevation.	35, 36, 42
	Inhalation	Sublethal	Chronic	Stable and unstable chromosome aberrations	45-47
	Inhalation	100 ppm	Chronic (work hours)	Leukopenia	60

TABLE III (CONTINUED)

Organism	Mode Exposure	Concentration of Exposure (ppm)	Duration of Exposure	Effect	Reference
Human	Inhalation	25 ppm	Chronic (work hours)	Lower hemoglobin levels, minor hematological deviations.	61
Dog	Inhalation	Sublethal	Acute	Hypertension and vasomotor paralysis	73
	Oral	Sublethal	Acute	Mucous membrane irritation, pulmonary edema and hemmorhage	73
Rat	Inhalation	20 ppm	6 hours/day 6 days/week 5 1/2 months	Delay in conditioned response time	81
	Inhalation	100 ppm	6 hours/day 5 days	Decreased incidence of spontaneous behavior	82
	Inhalation	450-500 ppm	5 hours/day 10 days	Increase in cytechrome P450 and aminopyrine demethylase activity	85
	Inhalation	Threshold	Acute	Disturbed oxidation-reduction and albumin production	79
(pregnant)	Inhalation	1-63.5 mg/m^3 (0.3125-19.84 ppm)	Chronic	Significant biochemical alterations in both pregnant female and fetus	87
	Oral	0.93 (0.71-1.23 g/kg)	Acute	LD_{50}	67
(young adult)	Oral	3.4 g/kg	Acute	LD_{50}	67
(older adult)	Oral	4.9 g/kg	Acute	LD_{50}	67

TABLE III (CONTINUED)

Organism	Mode of Exposure	Concentration of Exposure (ppm)	Duration of Exposure	Effect	Reference
Rat (older adult)	Oral	5.6 g/kg	Acute	LD_{50}	67
	Inhalation	44 ppm	7 hours/day 5 days/week	Leukopenia	72
	Inhalation	200 ppm	8 hours/day 5 days/week 90 days	Leukopenia	74
	Inhalation	1000 ppm	23.5 hours/ day, 105 hours	Body weight loss, nose and mouth hemorrhage, stomach distention, engorged blood vessels, reversal of polymorphonuclear: lymphocyte ratio	73
	Inhalation	88 ppm	204 days	Blood, bone marrow, spleen, and testes histopathological alterations	73
	Inhalation	40,000 ppm	20-35 minutes 5 exposures	3/8 male Long Evans rats died.	70
	Inhalation	10,000 ppm	12.5-30 minutes 1-17 days	2/10 male Long Evans rats died	70
	Inhalation	44 ppm	5 hours/day 4/days/wk 5-7 weeks	Slight leukopenia	72
Guinea pig	Inhalation	88 ppm	269 days	Blood, bone marrow, spleen, and testes histopathological alterations	73

TABLE III (CONCLUDED)

Organism	Mode of Exposure	Concentration of Exposure (ppm)	Duration of Exposure		Reference
Rabbit	Inhalation	80 ppm	243 days	Leukopenia and degeneration of seminiferous tubules	73
	Inhalation	35,000-45,000 ppm	3.7 minutes	Lightly anesthetized	75
	Inhalation	35,000-45,000 ppm	5 minutes	Excitation and tremors	75
	Inhalation	35,000-45,000 ppm	36 minutes	Death	75
	Inhalation	Sublethal	Chronic	Fatty bone marrow tissue, nucleic acid per unit weight of tissue decreased, synthesis of RNA and DNA increased	80
	Inhalation	10 mg/l (3,125 ppm)	6 hours/day 14 days	Hypertrophy of smooth endoplasmic reticulum ribosome loss, disappearance of segmental distention of ergastoplasm, swelling and myelin degeneration of mitochondria	86
	Inhalation	Sufficient to induce leukopenia		Reduced resistance to pneumonia and tuberculosis	88, 89, 91
	Injection	Sufficient to induce leukopenia		Active acute infections	90
	Inhalation	0.05 mg/l (15.625 ppm)	Chronic	Decreased phagocytic index and phagocytic number	92

3. Other Effects

Sunfish, Lepomis humilis, exposed to benzene concentrations of 35 to 37 ppm, died within one hour. Behavioral abnormalities, such as erratic movement, intoxication, and leaping out of the tank often preceded death. A concentration of 20 ppm benzene was lethal to 50 percent of a test population of bluegills, Lepomis machrochirus, within 24 to 48 hours. Behavioral observations included intermittent violent movement and distress at levels between 20 and 34 ppm. Based on the acute toxicity of benzene observed in these experiments, 6 ppm was calculated as a safe concentration (115).

Several species of freshwater fish were investigated for resistance to the acute toxicity of benzene by Pickering and Henderson (1966). Fathead minnows, Pimephales promelas, were slightly less susceptible to benzene toxicity in hard water. None of the other species were tested for toxicity variability due to mineral content of the water. A 24-hour TLm* of 35.5 ppm for soft water and 34.4 ppm for hard water was established for fathead minnows. Bluegills had a TLm of 22.5 ppm that did not vary for exposure intervals. Twenty-four hour benzene TLm's of 34.4 ppm and 36.6 ppm, respectively, were reported for goldfish and guppies (116). A compilation of reported effects due to acute benzene exposure for aquatic organisms is in Table IV.

---

*The concentration necessary to kill 50 percent of the test population within the specified time.

TABLE IV

REPORTED ACUTE BENZENE TOXICITY TO AQUATIC ANIMALS

ORGANISM	Concentration (ppm) resulting in death of all organisms within					Reference
	2 Hours	24 Hours	24 Hours	48 Hours	96 Hours	
Pimphales promelas (fathead minnow)	--	--	35.56	35.08	33.47	115
Lepomis macrochirus (bluegill)	--	--	22.49	22.49	22.49	115
Carassium auritus (goldfish)	--	--	34.42	34.42	34.42	115
Lebistes reticulatus (guppies)	--	--	36.60	35.60	36.60	115
Lepomis machrochiumus (bluegill)	60	34	--	--	--	114

Source: Modified from Assessing Potential Ocean Pollutants, National Academy of Sciences, Washington, D.C. 1975.

Chinook salmon, Oncorhynchus tsawytscha, and striped bass, Morone saxatilis, were subjected to sublethal benzene concentrations in freshwater and measured for exhibition of respiratory stress. Exposure to 6 ppm increased the respiration rates of juvenile chinook salmon, and these rates were 115 percent above normal 48 hours after exposure to 5 and 10 ppm. There was a 50 percent increase in the mean respiration of striped bass 24 hours after exposure to 5 ppm, but the total respiration increase was only 15 percent over an entire 96-hour exposure to 10 ppm. A narcosis and associated depression of respiration was evident among striped bass exposed for 48 hours to 5 ppm and 24 hours to 10 ppm, with recovery proceeding fairly rapidly after cessation of exposure. Doubling the current velocities in the respirometer produced a more rapid recovery by increased flushing of benzene from the gill filaments and increased oxygen availability (117). A summary of reported effects on aquatic organisms due to sublethal concentrations of benzene is contained in Table V.

The physiologic pathways followed by benzene in fish were described by Brocksen and Nailey (1973). Absorption occurs across the gill surface into the erythrocytes and lipoproteins of the blood, at which time it is transported to lipoid-containing tissues. Some of the benzene is excreted unaltered via the gills, while the remainder is metabolized to phenol in the liver, kidney, and various body tissues. The pathway appears to be the same as that for hydroxylation of sterols and pre-oxidation of lipids. These mechanisms or competition with lipid oxidation result in a buildup of benzene which is deposited

TABLE V

REPORTED SUBLETHAL BENZENE TOXICITY TO AQUATIC ANIMALS

ORGANISM	Concentration (ppm) at which Sublethal Effects Noted	Reported Effects	Reference
Oncorhynchus tshawytscha (chinook salmon)	5-10	Increased respiration rate	116
Morone saxatilis (striped bass)	5	Increased respiration rate in 24 hours	116
	5	Depressed respiration rate in 48 hours	116
	10	Respiration rate depressed in 24 hours	116
Pachygrapsus crassipes (lined shore crab)	-	Decreased chemotaxic response to food or sex hormones	113

Source: Modified from Assessing Potential Ocean Pollutants, National Academy of Sciences, Washington, D.C. 1975.

within the lipid-rich nervous system, and hence narcosis. It was postulated that the hexose monophosphate shunt in a fish with a low lipid content would not supply the $NADH_2$ necessary for enzymatic detoxification of benzene. Therefore, the compound would of necessity be catabolized to carbon dioxide and water, which is a slower process resulting in longer narcosis. Since the chinook salmon had fewer total body lipids (11%) than the striped bass (21%), this mechanism may account for the different respiratory responses observed (117).

Sublethal concentrations of benzene caused distinct chemoreception* abnormalities in marine crabs, while feeding behavior of the lined shore crab, Pachygrapsus crassipes, was inhibited by pure benzene as well as by an aqueous extract of crude petroleum containing the aromatic. The inhibition induced by benzene, however, tended to be more transient than that observed for the more complex crude extracts (114).

The ability of organisms to degrade or utilize benzene has been investigated. Pseudomonas and Achromobacter from soil have demonstrated the ability to use benzene as the sole carbon source for growth (107). Beijerinck found that benzene was not assimilated by Bacillus oligocarbophilus (108). However, it did decrease the light emissions, indicating reduced metabolism due to toxicity, from Vibrio luminosus, a luminous bacteria, at a level of 2,000 ppm (109).

---

*Sense organ or sensory nerve ending that is stimulated by a chemical substance.

Pathways for bacterial metabolic oxidation of the benzene ring have been discerned. Microorganisms convert benzene to catechols (dihydroxybenzenes) enzymatically before cleaving the ring and degrading it to CO_2 and water. Phenol occasionally appears as a metabolite, but catechols are typically the intermediate produced (110-113).

Photosynthetic-inhibiting effects of benzene on eight different tree species were studied and a permissible air pollution concentration of 0.1 mg/m^3 (0.031 ppm) was established. This level is defined as that which for a period of five minutes produced no noticeable photosynthetic decrease in any of the eight species (104). During observation of the giant kelp, Macrycystis pyrifera, no effect on photosynthetic activity was noted during a 96-hour exposure to 10 ppm benzene (105). In 1951, Currier examined the benzene distribution coefficient between water and paraffin oil and found an inverse relationship between compound solubility and toxicity. He concluded that an increased number of methyl groups on the benzene ring facilitated cellular penetration. Therefore, normal benzene toxicity to plants would be expected to be less than the toxicity of various substituted species of alkyl benzene.

C. AMBIENT DATA, POPULATION AT RISK AND MEASUREMENT TECHNOLOGY

The estimated normal concentration of ambient benzene in urban samples is reported to be 0.01 to 0.05 ppm (118). This level is not based on a vast compilation of data, since benzene is not routinely monitored in ambient air. In response to complaints during 1974, OSHA analyzed 269 industrial samples for the presence of benzene. However, a summary of the resultant levels detected is not readily available (119, 120).

Several reports of environmentally detected benzene at levels of 23 ppm or less are summarized in Table VI. These reports collectively provide evidence concerning the environmental presence of benzene. The analytical study by the U.S. Environmental Protection Agency of finished waters* extracted from the Mississippi River also examined the waste effluents of 60 industries that discharge into the river. A total of 53 organic chemicals, including acetone and toluene, were detected in 11 of the plants monitored. Although a trace of benzene was found in the finished waters, it was not detected in the effluent from any of the facilities, suggesting a source of contamination other than the industrial waste (121).

One research team, evaluating atmospheric benzene and toluene levels, took samples continuously at 17 sampling stations for 9 days. Quantitation of benzene levels revealed a maximum concentration of

*Water that has been processed by a purification and/or treatment plant.

TABLE VI

MONITORING DATA FOR BENZENE

Type of Sample	Geographical Location	Sampling* Method Used	Analysis* Technique	Quantities Detected	Reference
water and mud	Lower Tennessee River	CCE liquid-liquid extract	GC-MS	not reported	123
finished water	Carrollton Plant, New Orleans	CCE	preparative GC	not attempted	121
finished water	U.S. PHS Hospital Carville, La.			"trace" ppb-ppm range	124
"polluted" and "pure" drinking water	Prague, Czechoslovakia	inert gas stripping	GC, GC-MS	~0.1 ppb	126
ambient air	Vancouver, Canada	cold trap--GC column	rapid heating into GC	1-10 ppb	127
ambient air	vicinity of solvent reclamation plant	grab sample	direct injection into GC; MS, IR	23 ppm	128
ambient air	Los Angeles Basin	cold trap--firebrick	rapid heating into GC	.005-.022 ppm (V/V)	125

TABLE VI (CONCLUDED)

Type of Sample	Geographical Location	Sampling* Method Used	Analysis* Technique	Quantities Detected	Reference
ambient air	Downtown Los Angeles	grab sample	direct injection into GC	0.015-0.06 ppm (V/V)	129
ambient air	Los Angeles Basin	cold trap--glass beads	rapid heating into GC	average 0.015 ppm highest -.057 ppm (V/V)	130
ambient air	Zurich, Switzerland	charcoal trap--carbon disulfide extract	GC-MS GC-FI	0.054 ppm	131
ambient air	Riverside, Calif.	cold trap--GC column	GC-FI	0.007-0.008 ppm	132
ambient air	Toronto, Canada	cold trap--GC column	GC-FI	average 0.013 ppm, highest 0.098 ppm	122

Source: Howard, P. H., and P. R. Dustin, "Benzene Environmental Sources of Contamination, Ambient Levels, and Fate," Syracuse University Research Corporation, Syracuse, New York, December 1974.

*CCE--carbon chloroform extract; GC--gas chromatography; FI flame ionization; IR--infrared spectrometry; MS--mass

98 ppb, with an average concentration of 13 ppb. Based upon three factors, it was concluded that the benzene contamination of the air could be related to automobile emissions. The three key factors for this conclusion were: (1) the ratio of benzene to toluene; (2) the presence of distinct peak periods for both hydrocarbons at 0700, 1500, and 2100 hours; and (3) the relative concentrations detected at various sampling stations (122).

Determination of the risk to the general population is difficult using concentrations as low as those reported. If a cumulative effect exists, then the hazard may be significant. To estimate the risk generated by a production facility, a theoretical plant releasing benzene through ventilation and exhaust of internal air will be examined. Atmospheric dispersion from this plant would distribute the gaseous emissions downwind, as affected by the turbulent diffusion of the air. Hypothetical plant conditions can

be used as inputs to a Gaussian plume equation* from Turner's Workbook of Atmospheric Dispersion Estimates (133). The basic diffusion equation should be modified, however, due to the effect of the plant itself on the flow of air. Mechanical turbulence in the wake of a building tends to produce aerodynamic downwash, resulting in the fairly rapid diffusion of a gaseous emission down to the ground. This region of disturbed flow extends downwind a distance equal to several times the height of the building. While this situation does not result in a Gaussian distributed plume, it is proposed that a modification of the usual formula still can be employed. This is because the turbulent mixing in the wake of the building is assumed to be distributed uniformly in the vertical direction, analogous to the situation where a plume is trapped

---

*Ground level downwind concentrations resulting from a point source are predicted by the following equation:

$$X(x,0,0;H) = \frac{Q}{\pi \sigma_y \sigma_z u} \exp \left\{ -1/2 \left(\frac{H}{\sigma_z} \right)^2 \right\}$$

where: Q = uniform emission rate (grams/sec)
u = mean wind speed affecting the plume (meters/sec)
H = effective stack height; that is, sum of stack height plus plume rise (meters)
σ_y = horizontal dispersion coefficient evaluated in terms of downwind distance to the point for which the concentration is being computed (meters)
σ_z = vertical dispersion coefficient evaluated in terms of downwind distance to the point for which the concentration is being computed (meters)
$X(x,0,0;H)$ = the concentration in grams/meter^3 at a receptor located at ground level, x meters directly downwind from an emission source having an effective stack height of H meters.

below an inversion layer. A limited vertical mixing height can be modeled using the equation:

$$\chi(x,0,0;H) = \frac{Q}{\pi \sigma_y u\ (.8L)}$$

where L is the limit of the mixing depth. In the case of an isolated rectangular building it is assumed that L equals 1.5 times the building height.

In addition there is a horizontal wind turbulence which is assumed to result in an initial horizontal plume spread equal to the width of the building normal to the wind direction. This is analogous to an area source emission where the area source is the building top. This can be modeled using a further modification of the basic Gaussian diffusion equation. A virtual point source is assumed upwind from the building at a distance where the plume spread (for the given stability conditions) would equal the cross-wind width of the building. The modified diffusion equation is therefore:

$$\chi(x,0,0;H) = \frac{Q}{\pi \sigma'_y u\ (1.2h)}$$

where h is the building height and σ'_y is based on the downwind distance to the receptor point plus the upwind distance to the virtual point source.

Generally speaking, benzene production from petroleum feed-stocks is conducted in out-of-doors facilities with pressure-tight reaction and separation vessels. Thus, atmospheric release should

be virtually nil. However, as a most conservative approximation, assume that a hypothetical plant produces 185 million gallons of benzene annually, operating 360 days a year, 24 hours a day. Also assume that ten percent of this yield is lost in the form of flue gases, which are then treated by an emission control technique having a 98 percent efficiency. Additionally, assume that the building height is 50 feet (15.24 meters) and its width is 100 feet (30.48 meters). In this hypothetical example, the ambient benzene concentration 500 meters downwind of the plant is estimated to be 2.7 mg/m^3, or 0.845 ppm.* A downwind distance of 500 meters was chosen to represent a point within the perimeter of the production facility. Ambient concentrations in surrounding neighborhoods could be expected to be significantly lower. An individual positioned at this location would have to remain for only an instant to experience the brain electropotential enhancement which reportedly occurred at 2.8 mg/m^3 (39). The significance of this alteration in electropotential has not been determined. It does, however, indicate a predictable, measurable effect that may or may not be of importance to the exposed individual. Under these same assumed conditions, a person would have to remain positioned 500 meters downwind of the facilities for approximately 475 hours, or nearly 19.8 days, in order to inhale an amount equivalent to a worker exposed to the time-weighted average (10 ppm) during a

*Additional assumptions: (1) benzene is nonreactive; (2) the stability class is neutral; (3) the wind speed is 6 meters/second.

40-hour week recommended by the National Institute for Occupational Safety and Health (134).

The potential hazard constituted by this theoretical value is significant in comparison to the concentrations reported in the literature, as this hypothetical value is greater than all but the one level detected in the vicinity of a solvent reclamation plant. The degree of danger in relation to injury or disease is difficult to ascertain due to the lack of data correlating determined exposure levels with specific effects in the human population. If this situation were an actuality, it would be prudent to reduce the emissions, through use of controls and safety procedures, at least to the levels routinely found in urban areas. This reduction would not necessarily guarantee the safety of the surrounding population, however, it would ensure exposure levels comparable with those of the general populace. The primary risk associated with benzene would probably be to individuals employed by industries that utilize open-system procedures during either the production of benzene or the use of the chemical as an intermediate. More information pertaining to industrial benzene vapor levels is needed. This can be facilitated by sample collection coupled with benzene determination.

Various methodologies for collection of air samples have been developed. Both silica gel and activated charcoal have been successfully used as adsorbents for benzene in room temperature air. A prefilter of molecular sieves is advised for use with silica gel to

remove water vapor, as moisture can have a damaging effect on the ability of silica gel to adsorb benzene (135). The common use of activated charcoal eliminates this problem as the affinity of water for activated charcoal is not great. Therefore the use of activated charcoal, particularly in moist atmospheres, is generally accepted for the sampling of compounds that are not actively adsorbed on silica gel (136).

Other collection methods have included grab samples (137), bubblers (138), and absorption in scrubbers by nitrating solutions (139, 140). The use of plastic-film bags for collecting grab samples may result in loss of samples due to adsorption or permeation of the benzene vapor through the plastic. Absorption by scrubbing liquids is inconvenient for obtaining personal breathing-zone samples because several scrubbers must be connected in series in order to assure a high collection efficiency (134).

The concentration of benzene in an air sample using a cold trap, followed by direct gas chromatography injection, is the most sensitive, generally accepted, collection technique. However, with this method, only one sample is available for analysis, a distinct disadvantage. The charcoal or silica gel procedures followed by solvent extraction provide the analyst with the opportunity to aliquot the sample for repeated determinations, allowing replication and confirmation of experimental results. These procedures are commonly accepted and afford adequate sensitivity.

Extraction and/or preconcentration techniques for the detection of benzene in air or water samples have varied considerably (see Table VII). Both liquids and solids have been utilized as concentration adsorbents. However, the sensitivity increase facilitated by these techniques is accompanied by a longer sample work-up and processing time. Since increased handling of a sample increases the potential for inaccuracy, the addition of pre-deuterated benzene to the sample before analysis has been suggested (141). Establishment of the deuterated-to-undeuterated benzene ratio after concentration would allow a more accurate determination of the benzene contamination of the sample.

Analytical techniques for determining benzene in blood as well as air and water have been reported. Normally these procedures utilize a preconcentration step when low concentrations are to be determined, but a number of researchers have reported success using direct analysis. The use of laser Raman spectroscopy for detection and identification of as low as 20 ppm benzene in water has been enlisted due to its advantages of unnecessary sample preparation and system portability. Application of this technique is, however, limited to samples of high concentrations because of its low sensitivity (142, 143).

A continuous flow instrument has been devised for the determination of trace amounts of organics in chemical plant effluent based upon the ultraviolet adsorption of the aqueous solution. The

TABLE VII

SUMMATION OF TECHNIQUES FOR ANALYSIS OF BENZENE IN AIR AND WATER SAMPLES

Type of Sample	Adsorbent	Solvent	Qualitative and Quantitative Technique*	Limits of Detection	Reference
subsurface brines	--	isooctane extraction aqueous injection	UV absorption GC	-0.01	154
		Diethyl ether, chloroform, hexane extraction, chloroform or alcohol	GC GC-MS	--	123
drinking water	activated carbon	chloroform	Preparation GC, then infrared GC-MS	ppm-ppb range (not quantitative)	121 124
drinking water (New Orleans)	Activated carbon XAD resin liquid-liquid extraction, inert gas stripping (trap on GC column)	chloroform ethyl ether tetralin	GC, GC-MS GC, GC-MS Direct injection into GC, GC-MS	ppb range	158
air	methanol (with dry ice)	methanol	UV	accuracy below 10 ppm "poor"	159
air	silica gel	n-heptane	UV (not specific)	--	160
air	silica gel (personal air sampler)	ethanol	GC	ppm range	162

TABLE VII (CONTINUED)

Type of Sample	Adsorbent	Solvent	Qualitative and Quantitative Technique*	Limits of Detection	Reference
air	silica gel	iso-propyl alcohol and several other solvents	UV (not specific)	2-40 ppm	163
air	silica gel	--	indicator tube (cerium sulfate)--not specific	sensitivity--0.005 mg/l accuracy ±15%	164
air	charcoal	organic solvent	GC, GC-MS	20 ppm (10 l sample)	165
air	charcoal	carbon disulfide	GC-FI	20 ppm	169
air	charcoal	carbon disulfide	GC-FI	used down to 12 ppm	167
air	charcoal	carbon disulfide	GC-MS	50 ppb	131
air	firebrick (cold trap)	--	rapid heating into GC-FI	1 ppb ±25%	125
air	glass bead (cold trap)	--	rapid heating into GC-FI	1 ppb	130
air	GC packing (cold trap)	--	rapid heating into GC-EC, GC-FI	<10 ppb	127

TABLE VII (CONCLUDED)

Type of Sample	Adsorbent	Solvent	Qualitative and Quantitative Technique*	Limits of Detection	Reference
air	GC packing (cold trap)	--	rapid heating into GC-FI	--	161
air	GC packing (cold trap)	--	rapid heating into GC-FI	14.3 ±2.5 ppb	122
air	GC packing (cold trap)	--	rapid heating into GC-FI	0.1-0.2 ppb (100 ml sample)	132

Source: Adapted from P. H. Howard and R. R. Sustin, "Sources of Contamination, Ambient Levels, and Fate of Benzene in the Environment," Syracuse University Research Corporation, Syracuse, New York, December 1974.

*Ultraviolet spectrometry (UV); gas chromatography (GC); flame ionization (FI); electron capture detector (EC); mass spectrometry (MS).

light source used is a mercury discharge lamp (approximately 253.7 nm). This method is sensitive to 50 ppb benzene, but it is not specific for the hydrocarbon, since many other compounds also absorb UV light (144).

While evaluating the effect of 5000 ppm benzene on test animals, a technique utilizing direct injection of 1 μl of blood into a gas chromatograph was devised. Levels as low as 0.03 mg/100 ml (0.03 ppm) were detectable with this method (145).

Direct analysis of air samples has also been reported. Gas chromatography with direct injection (3 ml samples) was used to analyze ambient benzene in both air samples and in synthetic photo-chemical air. The sensitivity of a flame ionization (FI) detector was sufficient to detect individual aromatic hydrocarbons in the 0.05 to 1 ppm range in synthetic atmospheres and in the 0.005 to 4 ppm range in ambient air samples (129, 146).

Other analytical methods for the detection of benzene include direct ultraviolet spectrophotometry (147, 148, 149); colorimetry, which involves nitration followed by reaction with various ketones (138, 139, 149); gas chromatography (150-154); and direct estimation by means of colorimetric indicator tubes based on the colorimetric reaction between benzene and formaldehyde in the presence of sulfuric acid (155, 156).

For the analysis of benzene samples, gas chromatography is the method of greatest specificity and sensitivity and is widely

accepted. A wide variety of compounds interfere with both the colorimetric and direct spectrophotometric methods of analysis, as discussed previously. Colorimetric indicator tubes should be used only as a detection technique, not as a quantitative procedure (134).

Table VII contains a summary of adsorbents, solvents, and qualitative and quantitative techniques used.

D. SOURCES

As early as 1825, benzene was isolated by Faraday from a liquid condensate from compressed gas. The synthesis of this hydrocarbon first occurred in 1866 during chemical polymerization of acetylene performed by Berthelot.

Benzene has been commercially produced from coal since 1849, while production from petroleum began in 1941. It is still derived from both sources; however, 88 percent is now produced from petroleum (168). As of early 1975, the total U.S. production capacity for benzene as a chemical was 1,743 millic gallons per year (169). During the period from 1963 to 1973, the production capacity for benzene more than doubled. However, due to the derivation of benzene from petroleum products and the flux of the petroleum market, the estimation of the growth potential of benzene is difficult.

Only small amounts of benzene are present in crude oil. However, catalytic reforming of crude oil can increase benzene yields. Light oil is the major source of coal benzene. Crude light oil, which is 55 to 70 percent benzene is generated by the carbonization of coal to coke. Table VIII contains a listing of U.S. benzene producers, the source of their product (petroleum or coke), the location of their facilities, and the production capacities of these facilities. Figures 4 and 5 depict the geographic distribution and relative capacities of these facilities. From this map, it becomes apparent that the vast majority of the production plants are located

TABLE VIII

U.S. BENZENE PRODUCTION CAPACITY (1975)

PRODUCER	SOURCE[1]	LOCATION	CAPACITY[2] (millions of gallons)
Allied Chemical Corp.	P	Winnie, Tx.	3.0
Amerada Hess Corp.	P	St. Croix, Virgin Islands	15.0
American Petrofina	P	Port Arthur, Tx. Big Spring, Tx.	15.0 30.0
Armco Steel Corp.	C	Houston, Tx. Middletown, O.	0.9 2.0
Ashland Oil, Inc.	P	Ashland, Ky. North Tonawanda, N.Y.	50.0 15.0
Atlantic Richfield Co.	P	Houston, Tx. Wilmington, Calif.	44.0 16.0
Bethlehem Steel Corp.	C	Bethlehem, Pa. Lackawanna, N.Y. Sparrows Point, Md.	4.0 7.5 15.0
CF & I Steel	C	Pueblo, Colo.	3.0
The Charter Co.	P	Houston, Tx.	5.0
Cities Service Co., Inc.	P	Lake Charles, La.	25.0
Coastal States Gas Corp.	P	Corpus Christi, Tx.	70.0
Commonwealth Oil Refining Co., Inc.	P	Penuelos, P.R.	185.0
Crown Central Petroleum Corp.	P	Pasadena, Tx.	20.0
Dow Chemical, U.S.A.	P	Bay City, Mich.	30.0

TABLE VIII (CONTINUED)

PRODUCER	SOURCE	LOCATION	CAPACITY[2] (millions of gallons)
Exxon Corp.	P	Baton Rouge, La. Baytown, Tx.	72.0 62.0
Gulf Oil Corp.	P	Alliance, La. Philadelphia, Pa. Port Arthur, Tx.	70.0 33.0 38.0
Interlake, Inc.	C	Toledo, O.	1.0
Jones and Laughlin Industries, Inc.	P	Aliquippa, Pa.	10.0
Kerr-McGee Corp.	P	Corpus Christi, Tx.	8.0
Marathon Oil Co.	P	Texas City, Tx.	6.0
The Mead Corp.	C	Woodward, Ala.	1.4
Mobile Oil Corp.	P	Beaumont, Tx.	60.0
Monsanto Co.	P	Chocolate Bayou, Tx.	75.0
Northwest Industries, Inc.	C	Lone Star, Tx.	1.2
Pennzoil Co.	P	Shreveport, La.	12.0
Phillips Petroleum Co.	P	Sweeny, Tx.	22.0
Republic Steel Corp.	C	Youngstown, O.	4.0
Shell Chemical Co.	P	Deer Park, Tx. Odessa, Tx. Wilmington, Calif. Wood River, Ill.	75.0 5.0 20.0 40.0
Skelly Oil Co.	P	El Dorado, Kans.	14.0
Standard Oil Co. of California	P	El Segundo, Calif.	15.0

TABLE VIII (CONCLUDED)

PRODUCER	SOURCE[1]	LOCATION	CAPACITY[2] (millions of gallons)
Standard Oil Co. (Ind.)	P	Texas City, Tx.	85.0
Sun Oil Co.	P	Marcus Hook, Pa.	15.0
		Tulsa, Okla.	24.0
		Corpus Christi, Tx.	30.0
Tenneco, Inc.	P	Chalmette, La.	10.0
Texaco, Inc.	P	Port Arthur, Tx.	45.0
Union Carbide Corp.	P	Taft, La.	50.0
Union Oil Co. of California	P	Beaumont, Tx.	12.0
		Lemont, Ill.	33.0
Union Pacific Corp.	P	Corpus Christi, Tx.	10.0
United States Steel Corp.	C	Clairton, Pa.	40.0
		Geneva, Ut.	4.0
TOTAL			1,743.0

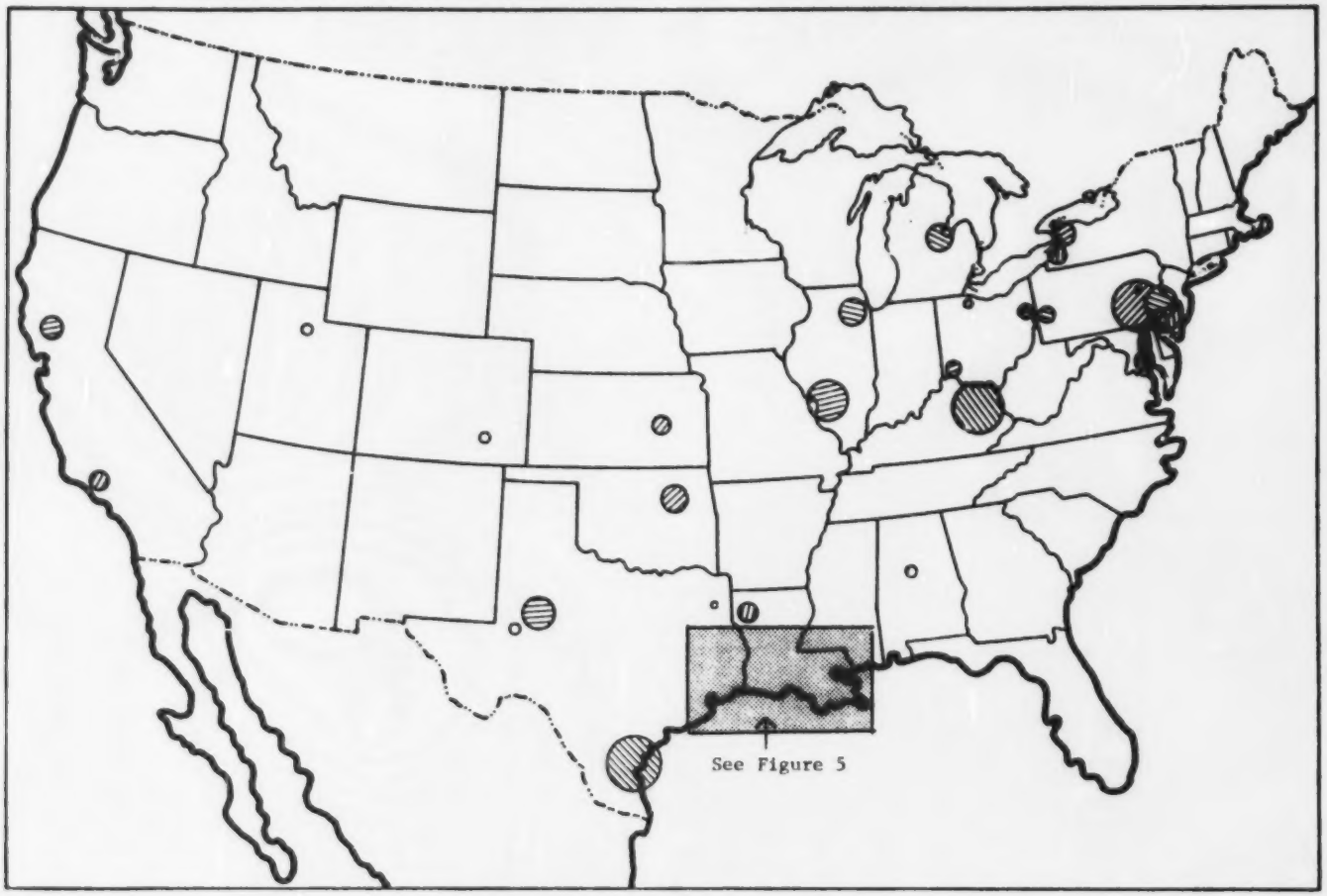
Source: 1975 Directory of Chemical Producers, United States of America, Chemical Information Services, Stanford Research Institute, Menlo Park, California.

[1] P = petroleum; C = coke

[2] As of January 1, 1975.

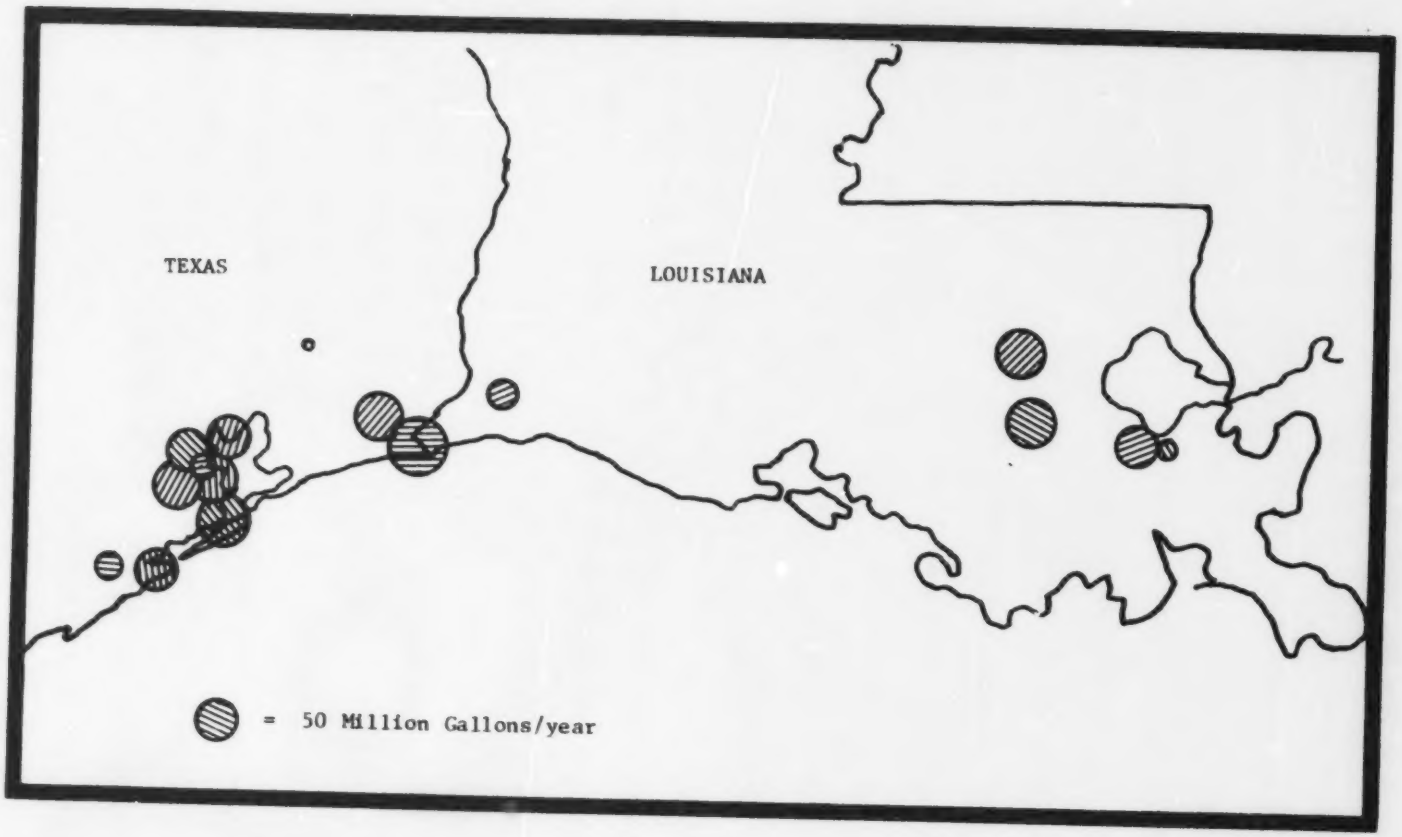
[3] Captive use.

FIGURE 4
GEOGRAPHIC DISTRIBUTION AND RELATIVE CAPACITIES OF BENZENE PRODUCTION FACILITIES



= 50 Million Gallons/year

FIGURE 5
DISTRIBUTION AND RELATIVE CAPACITIES OF BENZENE PRODUCTION
FACILITIES ON THE TEXAS AND LOUISIANA COASTS



along the Gulf of Mexico, particularly along the Texas coast. An additional source of benzene in the United States is importation. The U.S. International Trade Commission reports that approximately $110,000 worth of crude benzene was imported in 1974, while an average of 563 x 10^6 pounds of chemical benzene are imported per year (170). Extrapolation of this dollar value into a quantity is difficult due to the fluctuation of unit price for the crude compound.

Benzene, toluene, and xylene (BTX) can also be extracted from the liquid by-product of ethylene production by high severity pyrolysis. This mixture is stabilized by hydrotreating prior to the recovery of BTX aromatics by solvent extraction. The dissolved aromatics are separated from the solvent (di- and tri-ethylene glycol) by distillation, and the solvent-free aromatics are water washed and then separated into benzene, toluene, and xylene. The separated solvent is regenerated and recycled to the process, while the sludge produced is disposed of in landfills or by combustion (171).

In the past, benzene has also been produced by the hydrodealkylation of toluene, but the toluene is becoming less important as a source of benzene. Benzene produced by hydrodealkylation is more expensive than that obtained from catalytic reformate. Low demand for benzene and depressed prices have led to some production units being permanently retired during the last few years. Some dealkylation facilities will operate intermittently, depending upon benzene demand and the relative values of benzene and toluene (172).

Benzene is normally contained in steel tanks and shipped in railroad tank cars, 55-gallon returnable or one-way metal drums, tank trucks, and barges (173). Due to its volatility, it is possible that some environmental contamination occurs during production, storage, and transport, although the Compilation of Air Pollution Emission Factors published by the Environmental Protection Agency, states, "There are essentially no emissions from reforming operations," in the petroleum refining industry (174). The potential quantity lost is unknown; however, a reasonable assumption due to the economic incentive of the producer to limit losses is that no more than 1 percent of the total is lost. This theoretical loss to the environment, based on the 1975 production capacity, would be approximately 17.5 million gallons dispersed in a relatively specific geographical area.

Major stationary emissions sources for benzene do not appear to be production facilities, but those facilities wherein it is consumed as a synthetic intermediate. Additionally, major contamination sources are suspected to be oil spills, automotive, and coke oven emissions, since benzene is a component of the released hydrocarbons (190).

The primary commercial application of benzene is as a raw material for secondary synthesis (see Figures 6 and 7 and Table IX) (175). The efficiencies of the various synthetic processes utilized fluctuate from a yield of 100 percent during the hydrogenation of

Preceding page blank

Reproduced from best available copy.

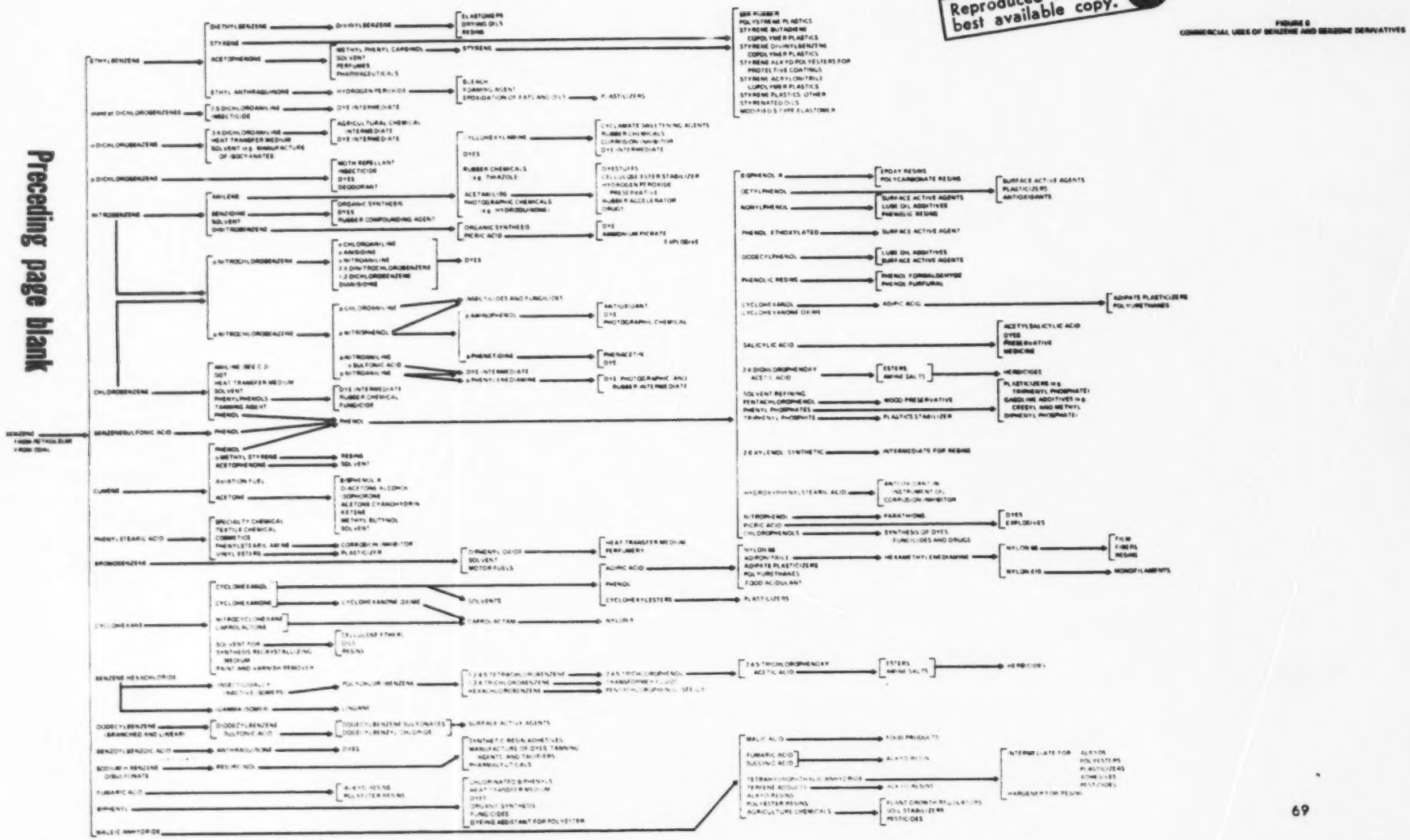
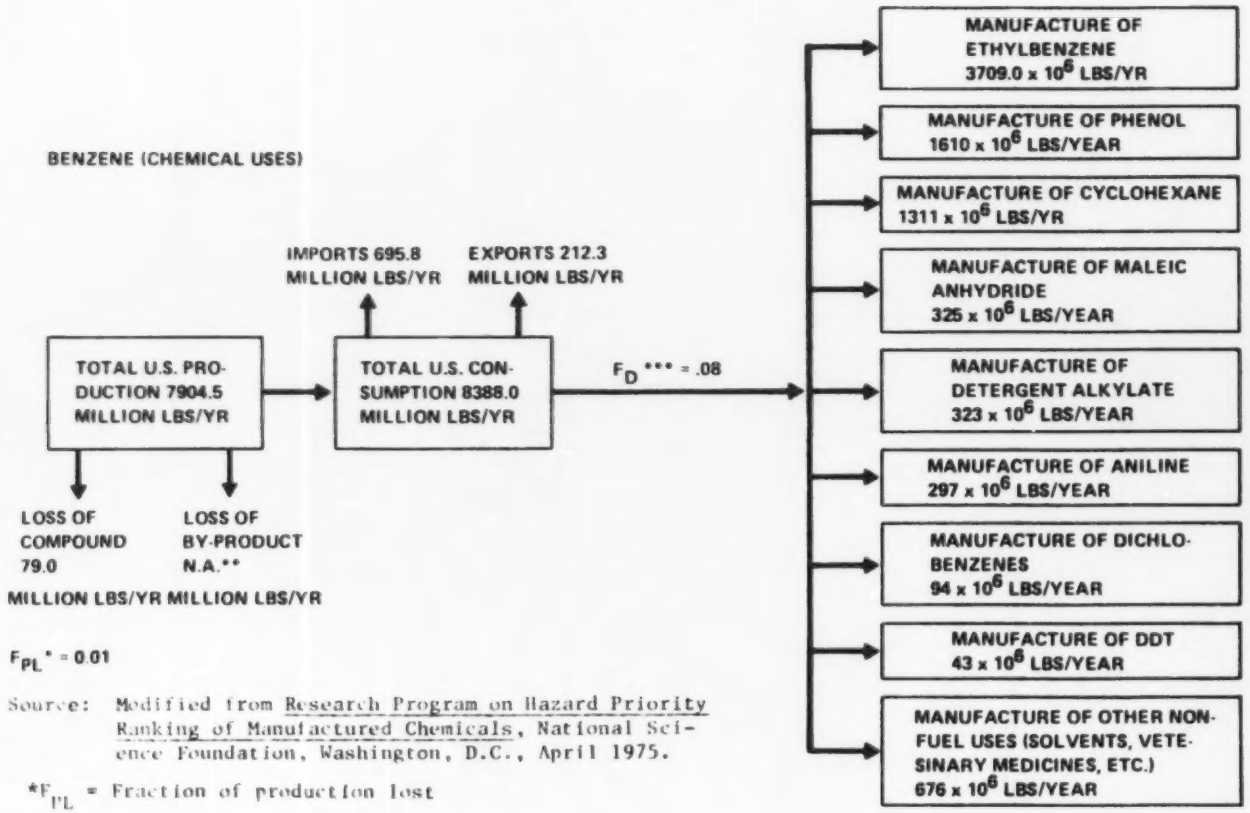


FIGURE 8
COMMERCIAL USES OF BENZENE AND BENZENE DERIVATIVES

FIGURE 7
THE FLOW OF BENZENE THROUGH THE UNITED STATES IN 1971


BENZENE (CHEMICAL USES)
IMPORTS 695.8 MILLION LBS/YR
EXPORTS 212.3 MILLION LBS/YR
TOTAL U.S. PRODUCTION 7904.5 MILLION LBS/YR
TOTAL U.S. CONSUMPTION 8388.0 MILLION LBS/YR
F_D *** = .08
LOSS OF COMPOUND 79.0 MILLION LBS/YR
LOSS OF BY-PRODUCT N.A.** MILLION LBS/YR
F_{PL}* = 0.01
MANUFACTURE OF ETHYLBENZENE 3709.0 x 10^6 LBS/YR
MANUFACTURE OF PHENOL 1610 x 10^6 LBS/YEAR
MANUFACTURE OF CYCLOHEXANE 1311 x 10^6 LBS/YR
MANUFACTURE OF MALEIC ANHYDRIDE 325 x 10^6 LBS/YEAR
MANUFACTURE OF DETERGENT ALKYLATE 323 x 10^6 LBS/YEAR
MANUFACTURE OF ANILINE 297 x 10^6 LBS/YEAR
MANUFACTURE OF DICHLOBENZENES 94 x 10^6 LBS/YEAR
MANUFACTURE OF DDT 43 x 10^6 LBS/YEAR
MANUFACTURE OF OTHER NON-FUEL USES (SOLVENTS, VETESINARY MEDICINES, ETC.) 676 x 10^6 LBS/YEAR

Source: Modified from Research Program on Hazard Priority Ranking of Manufactured Chemicals, National Science Foundation, Washington, D.C., April 1975.

*F_{PL} = Fraction of production lost

**N.A. = not available

***F_D = Fraction of dispersive use

Preceding page blank

TABLE IX

CONSUMPTION OF BENZENE[1]

Use	Percent
Manufacture of Ethylbenzene	44.2
Manufacture of Phenol	19.2
Manufacture of Cyclohexane	15.6
Manufacture of Maleic Anhydride	3.9
Manufacture of Detergent Alkylate	3.9
Manufacture of Aniline	3.5
Manufacture of Dichlorobenzenes	1.1
Manufacture of DDT	0.5
Manufacture of Other Non-Fuel Uses (solvents, veterinary medicine)	8.1

Source: Erskine, M. G., "Benzene," Chemical Economics Handbook, Stanford Research Institute, Menlo Park, California, 1972.

[1] 1971 estimate.

benzene to cyclohexane to an approximate yield of 57 percent during the oxidation of benzene to maleic acid (173). Figuring the difference between 100 percent and the reported synthetic yield, the upper limit of benzene potentially lost to the environment is calculated (see Table X) (175). These calculated losses are most likely much higher than those actually occurring due to the inclusion in the loss figure of quantities entering secondary synthetic steps rather than only those lost during the initial step involving benzene. In this calculation, benzene accounted for as lost may in fact have been converted into a byproduct or recycled. Thus, the loss figures reflect a most conservative upper bound. Since benzene is rarely used as a solvent, the last category in Table X, "other nonfuel uses," includes the consumption of benzene during synthesis of anthroquinone, chlorobenzene (other than for DDT, aniline, and phenol), benzene hexachloride, and diphenyl nitrobenzene (other than for phenol). The actual quantity in this "other nonfuel uses" category is determined by difference, and the losses would fluctuate depending upon the process employed (173). This category only amounts to eight percent of the total consumption; therefore, no attempt was made to calculate the loss.

As with losses incurred during production, losses during byproduct synthesis are not dispersed evenly. Table XI contains a compilation of the major byproducts producers accompanied by geographical location of the facilities. An asterisk indicates the

TABLE X

UPPER LIMIT OF THE AMOUNT OF BENZENE LOST FROM BYPRODUCT MANUFACTURING FACILITIES IN 1971

By-Product	Benzene Consumption (10^6 lbs)	(% of Total)	100% Minus % Yield *	Amount of Benzene Unaccounted for (10^6 lbs)
Ethylbenzene	3709	44	3	111
Phenol	1610	19	18	290
Cyclohexane	1311	15	0	0
Maleic Anhydride	325	4	43	140
Detergent Alkylate	323	4	20	65
Aniline	297	3	7	21
Dichlorobenzene	94	1	15	14
DDT	43	1	40	17
Other Nonfuel Uses	676	8	--	--
				658

Source: Benzene Environmental Sources of Contamination, Ambient Levels, and Fate, Life Sciences Division, Syracuse University Research Corporation, 1974.

*The yields used are for overall yields to the various by-products. Some of these processes (e. g., phenol) are more than one step and, therefore, the losses may be not only benzene but also the intermediate compounds (e. g., chlorobenzene or cumene with phenol production).

TABLE XI

LOCATION OF PLANTS UTILIZING BENZENE AS AN INTERMEDIATE
IN THE PRODUCTION OF SEVEN CHEMICALS

Company	Location	Product						
		Aniline	Cumene	Cyclohexane	Detergent Alkylate	Ethylbenzene	Maleic Anhydride	Phenol
Allied Chem. Corp.	Moundsville, W.V.	X					X	
American Cyanamid Co.	Bound Brook, N.J.	X						
	Willon Island, W.V.	X						
American Petrofina, Inc.	Big Spring, Tx.*			X		X		
Ashland Oil, Inc.	Ashland, Ky.*		X					
Baychem Corp.	New Martinsville, W.V.	X						
Clark Oil and Refining Co.	Blue Island, Ill.		X					
Coastal States Gas Prod. Co.	Corpus Christi, Tx.*		X					
Continental Oil Co.	Baltimore, Md.				X			
Cos-Mar, Inc.	Carville, La.					X		
Dow Chemical Co.	Freeport, Tx.					X		
	Midland, Mich.		X			X		X

TABLE XI (CONTINUED)

Company	Location	Product						
		Aniline	Cumene	Cyclohexane	Detergent Alkylate	Ethylbenzene	Maleic Anhydride	Phenol
E. I. DuPont de Nemours	Beaumont, Tx.	X						
	Gibbstown, N.J.	X						
El Paso Natural Gas Co.	Odessa, Texas					X		
First Mississippi Corp.	Pascagoula, Miss.	X						
Gulf Oil Corp.	Donaldsonville, Ga.					X		
	Philadelphia, Pa.*		X					
	Port Arthur, Tx.*		X	X				
Koppers Co., Inc.	Bridgeville, Pa.						X	
Marathon Oil Co.	Texas City, Tx.*		X					
Monsanto Co.	Alvin, Tx.*		X		X	X		
	St. Louis, Mo.						X	
Petro-Tex Chem. Corp.	Houston, Tx.						X	
Phillips Petroleum Co.	Sweeny, Tx.*			X				
	Guayana, P.R.*			X				
Reichold Chemicals, Inc.	Elizabeth, N.J.						X	
	Morris, Ill.						X	
								X

TABLE XI (CONTINUED)

Company	Location	Product						
		Aniline	Cumene	Cyclohexane	Detergent Alkylate	Ethylbenzene	Maleic Anhydride	Phenol
Rubicon Chemicals, Inc.	Geismar, La.	X						
Shell and Common. Chem., Inc.	Penuelas, P.R.			X				
Shell Oil Co.	Deer Park, Tx.*					X		
	Torrance, Calif.		X					
Sinclair-Koppers Co.	Port Arthur, Tx.					X		
Skelly Oil Co.	El Dorado, Kans.*		X					
Standard Oil Co. (Calif.)	Richmond, Calif.		X					
	El Segundo, Calif.*				X			
Standard Oil Co. (Ind.)	Texas City, Tx.*		X			X		
Standard Oil Co. (N.Y.)	Baytown, Tx.*			X				
Standard Oil Co. (Ohio)	Port Arthur, Tx.*				X			
Sun Oil Co.	Corpus Christi, Tx.*		X			X		

TABLE XI (CONCLUDED)

Company	Location	Product						
		Aniline	Cumene	Cyclohexane	Detergent Alkylate	Ethylbenzene	Maleic Anhydride	Phenol
Tenneco, Inc.	Fords, N.J.						X	
Texaco, Inc.	Port Arthur, Tx.*			X				
	Westville, N.J.*		X					
Union Carbide Corp.	Institute, W.V.				X			
	Seadrift, Tx.				X			
	Ponce, P.R.		X					
Union Oil Co. of California	Nederland, Tx.			X				
Union Pacific Corp.	Corpus Christi, Tx.*			X				
U.S. Steel Corp.	Neville Island, Pa.						X	
Witco Chem. Co., Inc.	Wilmington, Calif.				X			

Source: Erskine, M. G., "Benzene," Chemical Economics Handbook, Stanford Research Institute, Menlo Park, California, 1972.

*Location where the byproduct producer has a benzene production plant at the same site.

presence of a benzene manufacturer at the same location, which would indicate a considerably lower risk of loss to the environment during transport (173).

Comparison of Table VIII with Table XI indicates the byproduct facility locations to be primarily northeast, close to the consuming market, while the benzene feed stock locations are generally southwest, close to a petroleum source. Therefore, any significantly large loss during transport would occur along transportation routes between the Southwest and Northeast. The collision rate for tank cars has been calculated to be 1.52 collisions/10^6 miles (176); however, the quantity of benzene released in this manner has not been calculated.

Two significant sources of benzene contamination, not resulting from either commercial production or use, are oil spills and emissions from motor vehicles. The total oil influx into the ocean has been estimated to be 11-12,000 million pounds per year. This contamination arises from routine tanker discharges, accidents both in port and in free waters during oceanic exploration and production, discarded lubricants, pipeline breakage, incompletely burned fuel, and untreated industrial and domestic sewage (177). The benzene content of crude oil reportedly fluctuates from 0.001 to 0.4 percent (178). Assuming an average benzene concentration of 0.2 percent by weight in crude oil, between 22 and 24 million pounds of benzene are annually released into the oceans and waterways.

The contamination of the environment due to the benzene content of motor vehicle exhaust is more difficult to estimate. Evaporation of gasoline and exhaust emissions are both potential appreciable sources of loss. In terms of grams per vehicle-mile, approximately 20 percent of the urban transportation hydrocarbon emissions are due to evaporation, with the remainder due to exhaust (179). The total hydrocarbon emissions from transportation sources were estimated to be 29.4 billion pounds in 1971. This figure is approximately 55 percent of the total hydrocarbon emissions (180). The amount of benzene in fuel fluctuates with the grade used: regular--1.35 percent by weight; premium--0.81 percent by weight; American Petroleum Institute fuel--0.12 percent by weight (181).

Benzene, one of the more volatile gasoline constituents, increases and decreases in its relative proportion during evaporation depending upon the volatilities of all the components of the fuel (182). Presuming that benzene consistently comprises 1 percent of gasoline vapor and that gasoline vapor is 20 percent of the total annual vehicle emission, the benzene loss to the environment is 53.8 million pounds due to gasoline evaporation. Benzene is, by volume, 2.15 percent of the hydrocarbon emissions from a reciprocating engine (183). Because the lower weight hydrocarbons comprise more than 50 percent of the hydrocarbon emissions, the percentage of benzene, by weight, would be approximately 4 percent. Therefore, the

benzene emissions for 1971 from automotive exhaust would be 940.8 million pounds. The addition of the losses due to gasoline evaporation and motor vehicle exhaust yields an approximate value of 1 billion pounds, which would probably be geographically dispersed in direct proportion to population distribution.

Another possible source of environmental benzene contamination arises from the production of coke. According to the American Iron and Steel Institute Annual Statistical Report, 1974, 62,256,066 tons of coke were produced in 1974 (184). For each ton of coke produced, a yield of 3.2 gallons of light oil is realized, of which 1.85 gallons are benzene (185). Emissions of benzene from the coke ovens have not been reported (186, 187); however, emissions of hydrocarbon calculated as methane have been estimated for coke ovens with no emission controls (188). Assuming that the emitted hydrocarbons from coke ovens are of the same constitution as the light oil that is produced, and extrapolating the methane values to benzene, 1.96 pounds of benzene are emitted to the atmosphere for each ton of coke produced. Therefore, in 1974, approximately 122 million pounds of benzene were released to the air in the vicinity of coke ovens, which are located primarily in Alabama, Illinois, Indiana, Ohio, and Pennsylvania (189).

A summation of the benzene emissions from commercial production and use as well as from nonproduction sources is contained in Table XII. These are most likely the major routes of entry into

TABLE XII

BENZENE EMISSIONS TO THE ENVIRONMENT--1971
(IN MILLIONS OF POUNDS)

Source	Location	Quantity	Media of Entry	Controllability
Commercial benzene production, storage and transport	Primarily Southwest	79.0 (assumed 1% of total production)	Air Water	Point source, non-ducted, feasible
Commercial benzene usage	Primarily Southwest and Northeast	658 (based on reaction yields)	Air Water	Point and extended source. Nonducted, feasible except for solvent application.
Oil Spills	Oceans and Rivers	22-24	Water	Extended source. Non-ducted, infeasible
Motor Vehicle Emissions	Similar to population distribution.	1,000	Air	Extended source. Non-ducted, difficult.
Coke Ovens	Primarily, North Central States and Alabama	12? (1974)	Air	Point source, non-ducted, difficult

Source: Modified from P. H. Howard and P. R. Dustin, "Benzene--Environmental Sources of Contamination, Ambient Levels, and Fate," Syracuse University Research Corporation, Syracuse, New York, December 1974.

the environment; however, there may be other significant sources. As an example, the other 45 percent of the nontransportation hydrocarbon emissions sources may contain considerable concentrations of benzene, particularly fossil fuel combustion processes. The quantities involved are unknown and, therefore, emissions are incalculable (190).

Environmental transport of benzene from the point of emission has not been clearly defined. Projection of the distribution of benzene is possible utilizing the physical properties of high water solubility and vapor pressure (see Table I). Using these properties and assuming both constant mixing and less than saturation concentrations, the half-life of benzene was calculated to be 37 minutes in a square meter of water (191). Due to its relatively high solubility in water, however, atmospheric benzene would probably be reintroduced to the water via rainfall. Therefore, a continuous cycling between the air and water is likely to occur, indicating significant environmental mobility and possible dispersion and distribution ability.

E. CONTROL

The domestic production of benzene and its wide variety of uses result in the potential occupational exposure of 2 million people (192). Benzene emissions from production facilities can be controlled by using vapor condensation apparatus and installing local exhaust ventilation in major isolated process sections where benzene emissions occur. Although limited data are available, brief statements in medical reports indicate that there has been a significant decrease in benzene exposure levels since World War I, when concentrations were in the thousands of parts per million. By the late 1930's, levels had dropped to hundreds of ppm, and tens of ppm more recently (134). This would indicate that most industries are either meeting or within range of meeting the recommended exposure of not more than 10 ppm (32 milligrams/cubic meter of air) determined as a time-weighted average exposure for up to a 10-hour work day (134).

Several methods are available to control benzene emission from industrial sources:

a. Substitution of Process Materials or Equipment. If substitution of process materials or equipment is operationally and economically acceptable, this approach can be up to 100 percent effective in reducing benzene vapor exposures in industry. The effectiveness of this method has been demonstrated in the rubber coating industry (134).

However, before this control technique is applied, careful consideration must be given to the health hazards that may result from use of the new materials.

b. Proper Maintenance of Condensation Apparatus. The scrubbers and separators that are part of the catalytic reforming process that produces benzene from petroleum should be well maintained in order to eliminate the possibility of worker exposure to high levels of benzene resulting from leaks in the system, and to reduce the possibility of fire.

c. Process Ventilation and Process Location. Ventilation systems have several advantages over other control techniques. They are relatively inexpensive, easy to install and operate. For the control of benzene, spark-proof fans and systems can be designed and used to prevent the accumulation or recirculation of benzene vapors in work areas. Processes involving benzene should be isolated from other work areas, and entry to those areas should be restricted.

d. Proper Handling, Transport, and Good Housekeeping. The handling, transport, and storage of benzene, all present air quality control problems. Loading facilities should be equipped with a loading arm; a vapor collection adaptor; and pneumatic, hydraulic, or other mechanical means to force a vapor-tight seal between the adaptor and the hatch. When

loading through other than hatches, all loading and vapor lines should be equipped with vapor-tight connections that close automatically when disconnected. It is estimated that these precautions can reduce 55 to 60 percent of volatile organic emissions (such as benzene) from uncontrolled sources in gasoline marketing and other organic transfer operations (192).

Reduction of benzene emissions due to oil spills can only be effected by good housekeeping practices. These might be encouraged, however, by the levying of strict fines to offenders. In order to similarly reduce the hydrocarbon content in the atmosphere due to gasoline usage, only properly maintained automobiles appear as a feasible control. As a result of the activated carbon canisters currently installed on a motor vehicle crankcase and as the median model-year of automobiles in use increases, the total emissions of benzene as a result of crankcase fuel volatilization will decrease. This argument is supported by several sources (193, 194). For individual vehicles, light-duty crankcase and evaporative hydrocarbon emissions decreased by approximately 97 percent from pre-1961 automobiles to post-1972 automobiles (193). It is reasonable to assume that a fraction of these emissions will be benzene, and its reduction will closely follow total hydrocarbon reductions. Furthermore, non-methane hydrocarbon reductions of 63 percent from 1970 to 1980 are projected from a decrease in total automotive emission of about 72

percent for the same time period (194). Again, it is reasonable to assume that benzene emissions will closely track these overall figures.

Existing controls for coke oven operations are currently being expanded and updated. Controls for both the coke oven and larry car are being developed. The controls for the larry car consist of a totally enclosed environmentally controlled operator cabin, an automatic sequential lid-lifting mechanism, a drop-sleeve chute providing a seal with a charging hole, remote control operation ability of gravity hopper coal feed systems, a positive seal over the discharge hole, and a remote alignment system operation. Controls for the coke oven include a steam ejector or aspirating system to provide negative pressure within the oven during charging, improved lids having a modified design mating with an automatic mechanism on the larry car, and a double gas off-take relief piping to alleviate possible internal pressures caused by coal-oven roof blockage (195).

Table XII contains a summary of the primary sources of benzene emissions and the feasibility of control for each. A ducted (having a stack) point source will be the method of dispersion with the greatest feasibility of control, while the nonducted extended sources can be contained only by proper handling and operation techniques and adequate maintenance of the system involved.

REFERENCES

1. G. G. Hawley, The Condensed Chemical Dictionary, Van Nostrand Reinhold Co., New York, 100, 1971.

2. I. Sunshine, ed., CRC Handbook of Analytical Toxicology, The Chemical Rubber Company, Cleveland, Ohio, 604-605, 1969.

3. D. W. Fassett and D. I. Irish, eds., Industrial Hygiene and Toxicology, Second Revised Edition, Volume II, Toxicology. New York, 1220-1225, 1963.

4. G. W. Ayers and R. E. Muder, "Benzene," in Kirk-Othmer Encyclopedia of Chemical Technology, A. Stanton (ed.), 2nd Edition, John Wiley and Sons, Inc., N.Y., Vol. 3, 367-401, 1964.

5. C. A. MacKenzie, Unified Organic Chemistry, Harper and Row, New York, 91-97, 1962.

6. C. McAuliffe, "Solubility in water of C_1 - C9 hydrocarbons," Nature, 200, 4911, 1092-3, 1963.

7. A. Giacomelli and M. Spinetti, "Salt effects on the activity coefficients of benzene in aqueous solutions," Gazz. Chim. Ital., 102, 11, 965-973, 1972.

8. C. C. Lee, W. K. Craig, P. J. Smith, "Water soluble hydrocarbons from crude oil, "Bull. Envir. Contam Toxical., 12, 2, 212-217, 1974.

9. C. Sutton, J. A. Calder, "Solubility of higher molecular weight n-paraffins in distilled water and sea water," Envir. Sci. Tech., 8, 654, 1974.

10. American Society for Testing and Materials, D2359-69 in 1974 Annual Book of ASTM Standards, Part 29, 549, 1974.

11. American Society for Testing and Materials, D835-71 in 1973 Annual Book of ASTM Standards, Part 20, 347, 1973.

12. International Encyclopedia of Chemical Science, D. Van Nostrand and Company, 113-114, 1964.

13. W. A. Noyes, W. A. Mulac, and D. A. Harter, "Some aspects of the photochemistry of benzene," J. Chem. Phys., 44, 5, 2100-6, 1966.

Preceding page blank

14. M. Luria and G. Stein, "The Photoproduct of Benzene in Oxygenated Aqueous Solution," J. Chem. Soc., D, 1650, 1970.

15. L. Kaplan, L. A. Wendling, and K. E. Wilzback, "Photooxidation Aqueous Benzene. I. Identification of the Product as 1,3-cyclopentadiene-1-carboxaldehyde," J. Amer. Chem. Soc., 93, 15, 3819-20; 1971.

16. W. M. Jackson, J. L. Faris, and B. Donn, "Photolysis of Gaseous Benzene at 1470 Å," J. Phys. Chem., 71, 10, 3346-7; 1967.

17. J. Irina and K.
2537 Å," J. Chem. Soc., Chem. Commun., 19, 738, 1973.

18. A. P. Altshuller and J. J. Bufalini, "Photochemical Aspects of Air Pollution: A Review," Environ. Sci. Technol., 5, 39, 1971.

19. T. Matsuura and K. Omura, "Photochemical Hydroxylation of Aromatic Compounds," Synthesis, 3, 173-84, 1974.

20. J. Srbova, J. Teisinger, and S. Skramevsky, "absorption and elimination of inhaled benzene in man," Arch Industr Hyg., 2, 1-8, 1950.

21. J. Teisinger, V. Bergerova-Fiserova, and J. Kudrna, "The metabolism of benzene in man," (in Polish), Procovni lekarstvi 4, 175, 1952.

22. M. R. Duvcir, A. Fabre, L. Derobert, "The significance of benzene in the bone marrow in the course of benzene blood diseases," (in French) Arch Mal Prof 7, 77, 1946.

23. H. W. Gerarde, "The Aromatic Hydrocarbons," in Industrial Hygiene and Toxicology, Interscience Publishers and Sons, Inc., New York, 1220-25, 1963.

24. K. Nomiyama and H. Nomiyama, "Respiratory elimination of organic solvents in man: benzene, toluene, n-hexane, trichloroethylene, acetone, ethyl acetate, and ethyl alcohol," Int. Arch. Arbeitsmed., 32(1/2), 85-91, 1974.

25. "A new domestic poison," Lancet 1, 105, 1862.

26. C. Averill, "Benzole poisoning," Br. Med. J. 1, 709, 1889.

27. H. W. Gerarde, Toxicology and Biochemistry of Aromatic Hydrocarbons, Elsevier Publishing Co., New York, 97-108, 1965.

28. G. L. Conca and A. Maltagliati, "Transcutaneous absorption of benzene," (in Italian), Med. Lavoro 46, 194-98,1955.

29. F. Flury, "Moderne gewerbliche Vergiftungen in pharmakologisch-toxikologische Hinsicht," Naunym-Schmiedeberg's Arch. exp. Path. Pharmak. 138, 65-82, 1928.

30. L. Greenburg, "Benzol poisoning as an industrial hazard - I. The chemistry and industrial uses of benzol - II. Acute benzol poisoning," Public Health Reports 41, 1356-75, 1926.

31. "Benzene (Benzol). Inhalation of concentrated vapors may cause acute, chronic or fatal poisoning," Controlling Chemical Hazards Ser No. 6, U.S. Department of Labor, Div. Labor Standards, 1946.

32. A. Hamilton, "Benzene (benzol) poisoning," Arch Pathol II, 434-54, 1931.

33. E. Browning, Toxicity and Metabolism of Industrial Solvents, Elsevier Publishing Co., New York, 3-65, 1965.

34. C. L. Winek and W. D. Collom, "Benzene and toluene fatalities," J. Occup. Med., 13, 259-261, 1971.

35. L. Greenburg, M. R. Mayers, L. Goldwater and A. R. Smith, "Benzene (benzol) poisoning in the rotogravure printing industry in New York City," J. Ind. Hyg. Toxicol. 21, 395-420, 1939.

36. L. A. Erf and C. P. Phoades, "The hematological effects of benzene (benzol) poisoning," J. Ind. Hyg. Toxicol. 21, 421-35, 1939.

37. S. Imamiya, "The effects of hydrocarbons in human bodies in work environments," Kankojo Sozo 3, 62-69, September 1973.

38. I. S. Gusev, "Comparative toxicity studies of benzene, toluol, and xylol by the reflex activity method," in The Biological Effects and Hygienic Importance of Atmospheric Pollutants, Book 10, U.S.S.R. Literature on Air Pollution Occupational Diseases, Vol. 17, 60-67, 1968.

39. H. L. Hardy and H. B. Elkins, "Medical aspects of maximum allowable concentrations - Benzene," J. Ind. Hyg. Toxicol. 30, 196-200, 1948.

40. A. R. Smith, "Chronic benzol poisoning among women industrial workers - a study of the women exposed to benzol fumes in six factories," J. Ind. Hyg., 10, 73-93, 1928.

41. F. T. Hunter, "Chronic exposure to benzene (benzol) - II. The clinical effects," J. Ind. Hyg. Toxicol, 21, 331-54, 1939.

42. M. Savilahti, "More than 100 cases of benzene poisoning in a shoe factory," (in German) Arch Gewerbepathol. Gewerbehyg. 15, 147-57, 1956.

43. L. A. Zorina, and T. M. Sukharevskaya, "The mechanism of the development of the hemorrhagi syndrome in chronic benzene intoxications," (in Russian) Gigiena Gruda I Prof. Zabolevaniya, 10(8), 26-31, August 1966.

44. A. P. Rusinova, "Benzene and its homologues as poisons in electrical winding and insulation plants," U.S.S.R Literature on Air Pollution and Related Occupational Diseases, Vol. 7, 176-181, 1962.

45. A. M. Forni, A. Cappellini, E. Pacifico, and E. C. Vigliana, "Chromosome changes and their evolution in subjects with past exposure to benzene," Arch. Environ. Health, 23(5), 385-391, November 1971.

46. A. Forni, E. Pacifico, and A. Limonta, "Chromosome studies in workers exposed to benzene or toluene or both, "Arch. Environ. Health, 22, 373-378, 1971.

47. I. M. Tough and W. M. Court Brown, "Chromosome aberrations and exposure to ambient benzene," Lancet, 684, March 27, 1965.

48. A. Forni, "Chromosome changes due to chronic exposure to benzene" in Hygiene - Toxicology - Occupational Diseases, XVth International Congress of Labor Medicine, Wien 1966, Proceedings of the International Congress on Occupational Health, Vol. II,1, 437-439, 1966.

49. A. Forni and L. Moreo, "Cytogenic studies in a case of benzene leukaemia," Europ. J. Cancer, 3, 251-255, 1967.

50. A Forni and L. Moreo, "Chromosome studies in a case of benzene-induced erythroleukaemia," Europ. J. Cancer, 5, 459-463, 1969.

51. P. Delore and C. Borgomano, "Leucemia aigue au cours de l'intoxication benzenique. Sur l'origine toxique de certaines leucemies aigues et leurs relations avec les anemies graves," J. Med. Lyon, 9, 227-233, 1928.

52. T. B. Mallory, E. A. Gall, and W. J. Brickley, "Chronic exposure to benzene (benzol) - III. The pathologic results," J. Ind. Hyg. Toxicol. 21, 355-377, 1939.

53. E. C. Vigliani and G. Saita, "Benzene and leukemia," N. Engl. J. Med., 271, 872-876, 1964.

54. R. L. DeGowin, "Benzene exposure and aplastic anaemia followed by leukaemia 15 years later," J. Amer. Med. Ass., 185, 748-751, 1963.

55. E. M. Tareff, N. M. Kontchalovskaya, and L. A. Zorina, "Benzene leukemias," Acta. Un. Int. Cancer, 19, 751-755, 1963.

56. M. Aksoy, K. Dincol, S. Erdem, and G. Dincol, "Acute leukemia due to chronic exposure to benzene," Amer. J. Med. 52, 160-166, 1972.

57. H. Ludwig and A. Werthemann, "Benzo-Myelopathien," Schweig. med. Wschr., 92 378-384. 1962.

58. R. Girard and L. Revol, "La frequence d'une exposition benzenique au cours des hemopathies graves," Nouv. Rev. franc. Hemat, 10, 477-484, 1970.

59. T. Ishimaru, H. Okada, T. Tomiyasu, T. Tsuchimoto, T. Hoshino, and M. Ichimaru, "Occupational factors in the epidemiology of leukemia in Hiroshima and Nagasaki," Amer. J. Epidem, 93, 157-165, 1971.

60. L. Greenburg, "Benzol poisoning as an industrial hazard - VII. Results of medical examination and clinical tests made to discover early signs of benzol poisoning in exposed workers," Public Health Reports, 41, 1526-1539, 1926.

61. L. Butarewicz, S. Gosk, and M. Gluszczowa, "Examination of the state of health of women workers in the leather industry," (in Polish) Med. Przemyslowa, 20, 137-148, 1969.

62. H. Schrenk, W. P. Yant, S. J. Pearce, F. A. Patty, and R. R. Sayers, "Absorption, distribution and elimination of benzene by body tissues and fluids of dogs exposed to benzene," J. Ind, Hyg. Toxicol, 23, 20-34, 1941.

63. A. Sato, T. Nakajima, Y. Fujiwara, and K. Hirosawa, "Pharmacokinetics of benzene and toluene," Int. Arch. Arbeitsmed, 33(3), 169-182, 1974.

64. F. Schuckmann, "Possibilities of diagnosis and prophylactic measures against chronic benzene damage," (in German) Zbl. Arbeitsmed., 22(10), 294-300, 1972.

65. E. Browning, Toxicity and Metabolism of Industrial Solvents, Elsevier Publishing Co., New York, 3-65, 1965.

66. R. T. Williams, Detoxification Mechanisms, Wiley and Sons, New York, 188-194, 1959.

67. H. H. Cornish and R. C. Ryan, "Metabolism of benzene in nonfasted, fasted and aryl-hydroxylase inhibited rats," Toxicol, Appl. Pharmacol, 7, 767-771, 1965.

68. J. R. Mitchell, "Mechanism of benzene-induced aplastic anemia," Fed. Am. Soc. Exp. Biol., 30, 561, 1971.

69. Yu. Ye. Yakushevich, "Experimental data on hygienic assessment of continuous and intermittent action of benzene, toluol and xylene," (in Russian) Gigiena I. Sanit., No. 4, 6-10, 1973.

70. D. W. Furnas and C. H. Hine, "Neuotoxicity of some selected hydrocarbons," A.M.A. Arch. Industr. Hlth, 18, 9-15, 1958.

71. R. A. Wolf, V. K. Rowe, D. D. McCollister, R. L. Hollingsworth, and F. Oyen, "Toxicological studies of certain alkylated benzenes and benzene - Experiments in laboratory animals," Arch. Ind. Health, 14, 387-398, 1956.

72. W. B. Deichmann. W. E. MacDonald, and E. Bernal, "The hemopoietic tissue toxicity of benzene vaport," Toxicol. Appl. Pharmacol. 5, 201-224, 1963.

73. L. Dautrahande, Arch. Intern. Pharmacodynamie, 44, 394, 1933.

74. C. A. Nau, J. Neal, and M. Thornton, "C9-C12 fractions obtained from petroleum distillates - An evaluation of their potential toxicity," Arch. Environ. Health, 12, 382-393, 1966.

75. C. P. Carpenter, C. B. Shaffer, C. S. Weil, and H. F. Smyth, Jr., "Studies on the inhalation of 1:3 butadiene with a comparison of its narcotic effect with benzol, toluol, and styrene and a note on the elimination of styrene by the human," J. Ind. Hyg. Toxicol., 26, 69-78, 1944.

76. V. B. Dobrokhotov, "The mutagenic influence of benzene and toluene under experimental conditions," Gigiena I Sanit., 37(10), 36-39, October 1972.

77. I. S. Gusev, "The toxicity of low concentrations of aromatic hydrocarbons" in American Institute of Crop Ecology Survey of U.S.S.R. Air Pollution Literature, Vol. 15, 19-34, 1972.

78. I. S. Gusev, "A comparison of the effects of low concentrations of benzene, toluene and xylene," Hyg. Sanit., 32(2), 159-163, February 1967.

79. S. M. Pavlenko, "On several regularities of the effect of industrial non-electrolyte poisons taken into the organism along with water and air," Gigiena I Sanit., 37(1), 40-44, January 1972.

80. S. Nakamura and T. Nakajuma, "The effects of benzene on the nucleic acids in the bone marrow cells," Ind. Health, 5(3-4), 238-242, December 1967.

81. Y. V. Novikov, "Effect of small benzene concentrations on higher nervous activity of animals in chronic experiments," (in Russian) Gig. Sanit, 21, 20-25, 1956.

82. K. Horiuchi, S. Horiguchi, and S. Morioka, "Maximum allowable concentration of benzene in an animal experiment, "Osaka City Med. J., 13, 1-8, 1967.

83. Yu. V. Novikov, "Effect of small benzene concentrations on higher nervous activity of animals in chronic experiments, "Gig. Sanit. 2, 20-25, 1956.

84. J. Jonek, Z. Olknowski, and B. Zieleznik, "Histochemical studies on the spinal cord of mice poisoned with benzene, "Acta. Histochem., 20, 286-296, 1965.

85. K. Norpoth, U. Witting, M. Springorum, and C. Witting, "Induction of microsomal enzymes in the rat liver by inhalation of hydrocarbon solvents," Int. Arch. Arbeitsmed, 33(4), 315-321, 1974.

86. J. Jonek, M. Kaminski, H. K. Grzybek, and B. Panz, "Studies on ultrastructure of liver cells in rabbits in subacute benzene poisoning," (in POlish) Patol. Polska. 22(4), 641-651, 1971.

87. V. A. Gofmekler, N. N. Pushkina, and G. N. Klevtsova, "Some biochemical aspects of the embryotropic effect of benzene and formaldehyde," Hyg. Sanit. 33(7-9), 112-116, July-September 1968.

88. M. C. Winternitz and A. D. Hirschfelder, "Studies upon experimental pneumonia in rabbits-Parts I - III," J. Exp. Med. 18, 657-665, 1913.

89. B. S. Kline and M. C. Winternitz, "Studies upon experimental pneumonia in rabbits - V. The role of the leucocyte in experimental pneumonia. The relation of the number of organisma injected to the mortality," J. Exp. Med., 18, 50-60, 1913.

90. H. G. Weiskotten and H. S. Steensland, "Action to benzol-IV. Spontaneous infections with special reference to the diphasic leucopenia (rabbit), "Exp. Res., 37, 215-223, 1917.

91. W. C. White and A. M. Gammon, "The influence of benzol inhalations on experimental pulmonary tuberculosis in rabbits, "Trans. Assocl. Amer. Phys., 29, 332-337, 1914.

92. A. P. Volkora, "Effect of chronic benzene intoxication on the phagocytic activity of rabbits, "Gig. Sanit., 24(1), 80-82, 1959.

93. M. Kissling and B. Speck, "Chromosome aberrations in experimental benzene intoxication. "Helv. med. Acta., Vol. 36, 59-66, 1971.

94. H. Desoille, M. Philbert and C. Albahary, "Hormonal influences in chronic benzene intoxication in guinea pigs. Influence of gestation on white and red blood cell counts in guinea pigs with and without moderate benzene intoxication during the entire gestation period," (in French) Arch. Mal. Prof., 28, 329-339, 1967.

95. E. T. Kimura, D. M. Ebert, and P. W. Dodge, "Acute toxicity and limits of solvents residue for sixteen organic solvents," Toxicol. appl. Pharmacol. 19, 699-704, 1971.

96. S. Harashina, H. Sakurai and K. Nakamura, "Difference in responses of two strains of mice to benzene inhalation," (in Japanese) Japan J. Ind. Health, 7(11), 8-12, December 1965.

97. R. W. Baldwin, H. C. Palmer, R. T. Parfitt, M. W. Partridge, H. J. Vipond, and J. A. Waite, "Studies on the carcinogenicity of trycycloquinazoline," A. R. Brit, Emp. Cancer Campgn., 39, 414-420, 1961.

98. M. M. Coombs and C. J. Croft, "Carcinogenic derivatives of cyclopenta(a)phenanthrene," Nature (lond.), 210, 1281-1282, 1966.

99. O. D. Laebrum, "Reticulum cell neoplasms in normal and benzene treated hairless mice," Acta. Path. Microbial. Scand., Sect. A, 81, 57-63, 1973.

100. G. O. E. Lignac, "Die Benzolleukamie bei Menschen und weissen Mausen. III. Zweite Benzolversuchsreiche-von 54 Mausen gehen 8 an Leukamie oder Lymphoplastoma infiltrans aleucaemicum zugrundefruhere Teerbenzolversuchz," Krankheitsforsch, 9, 426-453, 1932.

101. K. Hiraki, S. Irino, and I. Miyoshi, "Development of subcutaneous sarcomas in Swiss mice given repeated injections of benzene in olive oil," Gann, 54, 427-431, 1963.

102. A Kirschbaum and L. C. Strong, "Influence of carcinogens on the age incidence of leukaemia in the high leukaemia F strain of mice," Cancer Res., 2, 841-845, 1942.

103. J. L. Amiel, "Essai negatif d'induction de leucemies chez les souris par le benzene," Rev. franc. Etud. Clin. Biol., 5, 198-199, 1960.

104. V. S. Nikolayevskiy and A. T. Miroshnikova, "The air pollution levels permissible for plants," (in Russian) Gigiena I. Sanit. No. 4, 15-18, 1974.

105. W. J. North, K. A. Clendenning, and H. L. Scotten, "The Effects of Waste Discharges Upon Kelp," Quarterly Progress Report, 4th quarter, IMR Ref. 60-10, Institute of Marine Resources, Univ. of California, La Jolla, California, 1959.

106. H. B. Currier, "Herbicidal properties of benzene and certain methyl derivatives," Hilgardia 20 = 383, 1951.

107. D. T. Gibson and W. K. Yeh, "Microbial degradation of aromatic hydrocarbons," in Proceedings of the Microbial Degradation of Oil Pollutants Workshop, December 4-6, 1972, Georgia State University, LSU-SG-73-01, 33-38, 1972.

108. K. Lantzsch, "Actinomyces oligocarbophilus (Bacillus oligocarbophilus Beij.) Its change of form and its physiology," Zent. Bakteriol. Parasitenk. Abt. II, Vol. 57, 390-319, 1922.

109. F. Koessler and I. Wolburg, "Metabolism of microorganisms as indicators for toxic substances, (in German) Z. Ges. Hyg., 17(5), 317-321, May 1971.

110. D. T. Gibson, "Initial Reactions in the Degradation of Aromatic Hydrocarbons," in Degradation of Synthetic Organic Molecules in the Biosphere: Natural, Pesticidal and Various Other Man-made Compounds, National Academy of Sciences, Washington, D.C., 116-136, 1972.

111. E. K. Marr and R. W. Stone, "Bacterial Oxidation of Benzene," J. Bacteriol. 81:425-430, 1961.

112. R. J. O'Connor, B. W. Weinrich, and W. A. Darlington, "Phenol and the Microbiological Conversion of Benzene to Catechol," Bacteriol. Proc. 1964:104-105, 1964.

113. D. T. Gibson, J. R. Koch, and R. E. Kallio, "Oxidative Degradation of Aromatic Hydrocarbons by Microorganisms. I. Enzymatic Formation of Catechol from Benzene," Biochemistry 7(7): 2653-2662, 1968.

114. J. S. Kittredge, "Effects of the Water-soluble Component of Oil Pollution on Chemoreception by Crabs," NTIS Pub. No. AD 738505, Division of Neurosciences, City of Hope National Medical Center, Duarte, California, 1971.

115. H. Turnbull, J. G. DeMann, and R. F. Weston, "Toxicity of Various Refinery Materials to Fresh Water Fish," Ind. Eng. Chem., 46, 324-333, 1954.

116. Q. H. Pickering and C. Henderson, "Acute Toxicity of Some Important Petrochemicals to Fish," J. Water Pollut. Control Fed., 38, 1419-1429, 1966.

117. R. W. Brocksen and H. T. Bailey, "Respiratory Response of Juvenile Chinook Salmon and Striped Bass Exposed to Benzene, a water-soluble component of crude oil," in Proceedings of Joint Conference on Prevention and Control of Oil Spills, March 13-15, American Petroleum Institute, Washington, D.C., 783-791, 1973.

118. A. P. Altshuller, Atmosphere Analysis by Gas Chromatography, U.S. Dept. of Health, Education, and Welfare, Public Health Service, Division of Air Pollution, 1969, and California Dept. of Public Health, SDPH1-SDPH1-50, August 1966.

119. Dr. Russell Heinricks of OSHA, personal communication, August 1975.

120. Floyd Madsen of OSHA, personal communication, August 1975.

121. U.S. Environment Protection Agency, "Industrial Pollution of the Lower Mississippi River in Louisiana," Region VI, Dallas, Texas, Surveillance and Analysis Division, 1972.

122. S. Pilar and W. F. Graydon, "Benzene and toluene distribution in Toronto atmosphere," Environmental Science and Technology, 7/7, 628-631, July 1973.

123 M. Gordon and P. C. Goodley, "Isolation and Characterization of Industrial Organic Pollutants in Water," Amer. Chem. Soc., Div. Water, Air Waste Chem., Gen. Pap., 11, 1, 91-4, 1971.

124. J. J. Friloux, "Petrochemical Wastes as a Water Pollution Problem in The Lower Mississippi River," Paper Submitted to the Senate Subcommittee on Air and Water Pollution, April 5, 1971.

125. R. E. Neligan, M. J. Leonard, and R. J. Bryan, "The Gas Chromatographic Determination of Aromatic Hydrocarbons in the Atmosphere," ACS Nat. Meeting, September 12-17, Atlantic City, N.J., 1965.

126. J. Novak, J. Zluticky, V. Kubelka, and J. Mostecky, "Analysis of Organic Constituents Present in Drinking Water," J. Chromatogr., 76, 1, 47-50, 1973.

127. I. H. Williams, "Gas Chromatographic Techniques for the Identification of Low Concentrations of Atmospheric Pollutants," Anal. Chem., 37, 13, 1723-32, 1965.

128. J. C. Smoyer, D. E. Shaffer, and I. L. DeWitt, "Program to sample and analyze air pollution in the vicinity of a chemical reclamation plant," Inst. Environ. Sci., Tech. Meet., Proc., 17, 339-45, 1971.

129. A. P. Altshuller and T. A. Bellar, "Gas Chromatographic Analysis of Hydrocarbons in the Los Angeles Atmosphere," J. Air Pollution Control Assoc., 13, 2, 81-7, 1963.

130. W. A. Lonneman, T. A. Bellar, and A. P. Altshuller, "Aromatic hydrocarbon in the atmosphere of the Los Angeles Basin," Environ. Sci. Technol., 2, 11, 1017-20, 1968.

131. K. Grob and G. Grob, "Gas-liquid Chromatographic-mass Spectrometric Investigation of C_6-C_{20} Organic Compounds in an Urban Atmosphere. Application of ultratrace analysis on capillary columns," J. Chromatogr., 62, 1, 1-13, 1971.

132. E. R. Stephens, "Hydrocarbons in Polluted Air," Summary Report, Coordinating Research Council Project CAPA-5-68, Statewide Air Pollution Research Center, Univ. of Calif., Riverside, U.S. Nat. Tech. Inform. Serv., PB Rept. 230-993, 1973.

133. D. B. Turner, Workbook of Atmospheric Dispersion Estimates, U.S. Dept. of Health, Education, and Welfare, Revised, 1969.

134. National Institute for Occupational Safety and Health, U.S. Dept. Of Health, Education, and Welfare, Criteria for a Recommended Standard...Occupational Exposure to Benzene, HEW Publication No. (NIOSH) 74-137, 1974.

135. N. E. Whitman and A. E. Johnston, "Sampling and anaylsis of aromatic hydrocarbon vapors in air: A gas-liquid chromatographic method," Amer. Ind. Hyg. Assoc. J., 25, 5, 464-9, 1964.

136. H. Buchwald, "Activated silica gel as an adsorbent for atmospheric contaminants," Occupational Health Rev., 17, 4, 14-18, 1965.

137. B. S. Smith, and J. O. Pierce, "The use of plastic bags for industrial air sampling," Am. Ind. Hyg. Assoc. J. 31, 343-48, 1970.

138. R. G. Bond and C. P. Straub, Handbook of Environmental Control, Volume I: Air Pollution, 108, 1972.

139. B. H. Dolin, "Determination of benzene: Detection and estimation of benzene in the presence of toluene, xylene, and other substances," Ind. Eng. Chem. Anal. Ed. 15, 242-47, 1943.

140. B. S. Levine (ed), "Quantitative determination of benzene in the air," USSR Literature on Air Pollution and Related Occupational Diseases 8, 47-51, 1963.

141. S. P. Wasik and W. Tsand, "Determination of trace amounts of contaminants in water by isotope dilution gas chromatography," Anal. Chem., 42, 13, 1649-51, 1970.

142. E. B. Bradley and C. A. Frenzel, "Exploitation of laser raman spectroscopy for detection and identification of molecular water pollutants," Water Res. 4, 1, 125-8, 1970.

143. E. G. Bradley, C. A. Frenzel, J. Reeves, R. McConnell, and K. Lane, "Detection and identification of molecular water pollutants by lasar raman spectroscopy," U.S. Nat. Tech. Inform. Serv., PB Rept. 208029, 1972.

144. H. C. Bramer, M. J. Walsh, and S. C. Caruso, "Instrument for monitoring trace organic compounds in water," Water Sewage Works, 113, 8, 275-8, 1966.

145. S. Fati, "Gas chromatography in the detection of some industrial poisons in blood," Int. Symp. Chromatogr. Electrophoresis, 5th 1968, Ann Arbor-Humphrey Sci. Publ., Inc., 383-93, 1969.

146. A. P. Altshuller and C. A. Clemons, "Gas chromatographic analysis of aromatic hydrocarbons at atmospheric concentrations using flame ionization detection," Anal. Chem., 34, 466, 1962.

147. H. B. Elkins, L. D. Pagnotto and E. M. Comproni, "The ultraviolet spectrophotometric determination of benzene in air samples adsorbed on silica gel," Anal. Chem. 34, 1797-1801, 1962.

148. P. A. Maffett, T. F. Doherty and J. L. Monkman, "A direct method for the collection and determination of micro amounts of benzene or toluene in air," Am. Ind. Hyg. Assoc. Quate. 17, 186-88, 1956.

149. Analytical Abstracts Committee, Analytical Abstracts, Benzene, American Industrial Hygiene Association, 1965.

150. C. L. Fraust, and E. R. Hermann, "Charcoal sampling tubes for organic vapor analysis by gas chromatography," Am. Ind. Hyg. Assoc. J. 27, 68-74, 1966.

151. F. H. Reid and W. R. Halpin, "Determination of halogenated and aromatic hydrocarbons in air by charcoal tube and gas chromatography," Am. Ind. Hyg. Assoc. J. 29, 390-96, 1968.

152. R. J. Sherwood and F. W. G. Carter, "The measurement of occupational exposure to benzene vapour," Ann. Occup. Hyg. 13, 125-46, 1970.

153. R. Levadie and J. F. Harwood, "An application of gas chromatography to analysis of solvent vapors in industrial air," Am. Ind. Hyg. Assoc. J. 21, 20-24, 1960

154. N. E. Whitman and A. E. Jonston, "Sampling and analysis of aromatic hydrocarbon vapors in air: A gas-liquid chromatographic method," Am. Ind. Hyg. Associ. J. 25, 464-69, 1964.

155. P. Kaljkowsky, "Indicator-tube method for the determination of benzene in air," Analyst 94, 918-20, 1969.

156. R. M. Ash and J. R. Lynch, "The evaluation of gas detector tube systems--Benzene," Am. Ind. Hyg. Assoc. J. 32, 410-11, 1971.

157. W. M. Zarrella, R. J. Mousseau, N. D. Coggeshall, M. S. Norris, and G. J. Schrayer, "Analysis and significance of hydrocarbons in sub-surface Brines," Geochim, Cosmochim Acta, 31, 7, 1155-66, 1967.

158. U.S. Environmental Protection Agency, "Draft Analytical Report: New Orleans Area Water Supply Study," Region VI, Surveillance and Analysis Division, Lower Mississippi River Facility, 1974.

159. T. Dambrauskas and W. A. Cook, "Methanol as the absorbing reagent in the determination of benzene, toluene, xylene, and their mixtures in air," Am. Ind. Hyg. Assoc. J. 24, 6, 568-75, 1963.

160. R. Bencze, "A rapid spectrophotometric method for the simultaneous determination of cumene and benzene in air," Z. Anal. Chem., 211, 6, 422-9, 1965.

161. M. Selucky, J. Novak, and J. Janak, "use of the chromatography equilibration procedure for air pollution studies. Determination of minute amounts of benzene, chlorobenzene, and nitrobenzene in air," J. Chromatogr., 28, 2, 285-92, 1967.

162. R. J. Sherwood, "Monitoring of benzene exposure by air sampling," Amer. Ind. Hyg. Ass., J., 32, 12, 840-6, 1971.

163. H. B. Elkins, L. D. Pagnotto, and E. M. Comproni, "The ultraviolet spectrophotometric determination of benzene in air samples adsorbed on silica gel," Anal. Chem., 34, 1797-801, 1962.

164. P. G. Koljkowsky, "Indicator-tube method for the determination of benzene in air," Analyst, 94, 1123, 918-20, 1969.

165. C. V. Cooper, L. D. Whites and R. E. Kupel, "Qualitative detection limits for specific compounds utilizing gas chromatographic fractions activated charcoal, and a mass spectrometer," Amer. Ind. Hyg. Ass., J., 32, 6, 383-6, 1971.

166. R. M. Ash and J. R. Lynch, "Evaluation of gas detector tube systems benzene," Amer. Ind. Hyg. Ass., J., 32, 6, 410-11, 1971.

167. L. D. White, D. G. Taylor, P. A. Mauer, and R. E. Kupel, "Convenient optimized method for the analysis of selected solvent vapors in the industrial atmosphere," Amer. Ind. Hyg. Ass., J., 31, 2, 225-32, 1970.

168. G. W. Ayers and R. E. Muder, "Benzene," Encyclopedia of Chemical Technology, 2nd ed., pgs. 367-401, 1964.

169. Chemical Information Services, Stanford Research Institute, 1975 Directory of Chemical Producers - U.S.A., 1975.

170. U.S. Trade Commission, May 1975.

171. U.S. Environmental Protection Agency, Development Document for Proposed Effluent Limitations Guidelines and New Source Peformance Standards for the Major Organic Products Segment of the Organic Chemicals Manufacturing Point Source Category, EPA 440/1-73/009, 82-85, December 1973.

172. M. G. Erskine, "Toluene," Chemical Economics Handbook, March 1972.

173. M. G. Erskine, "Benzene," Chem. Econ. Handbook, S.R.I., 618.5021A-618.5024G, 1972.

174. Office of Air and Water Programs, U.S. Environmental Protection Agency, Compilation of Air Pollutant Emission Factors, 2nd ed, April 1973.

175. Chemical Origins and Markets, Stanford Research Institute 18-19, 1967.

176. J. M. Lippian, "The transportation of hazardous materials: Transport of benzene by tank car," U.S. Nat. Tech. Inform. Service, AD Rept. 771105, 1973.

177. M. Blumer; H. L. Sanders; J. F. Grassle, and G. R. Hampson, "A small oil spill," Environment, 13, 2, 2, 1971.

178. A. D. Green and C. E. Morrell, "Petroleum Chemicals," in Kirk-Othmer Encyclopedia of Chemical Technology, Vol. 10, 1st Edit., Interscience Publ. Inc., N. Y., pp. 177-210, 1973.

179. R. E. Wendell; J. E. Norco, and K. G. Croke, "Emission prediction and control strategy: Evaluation of pollution from transportation systems," J. Air Pollut. Contr. Assoc., 23, 91, 1973.

180. Council on Environmental Quality, Environmental Quality-The Fourth Annual Report of the Council on Environmental Quality, P266, Government Printing Office, Washington, D. C., 1973.

181. W. N. Sanders and J. B. Maynard, "Capillary gas chromatographic method for determining the C_3-C_{12} hydrocarbons in full-range motor gasolines," Anal. Chem., 40, 3, 527-35, 1968.

182. B. J. Mair, "Hydrocarbons isolated from petroleum," Oil and Gas J., 62, 37, Sept. 14, 130-134, 1964.

183. K. Schofield, "Problems with flame ionization detectors in automotive exhaust hydrocarbon measurement," Environ. Sci. Technol., 8, 9, 826-834, 1974.

184. American Iron and Steel Institute, Annual Statistical Report, 1974, Washington, D.C., 1975.

185. W. L. Faith, D. B. Keyes and R. L. Clark, Industrial Chemicals, 125-127, 1965.

186. Personal communication Ms. Paul Caplan, Occupational Safety and Health Administration, Cincinnati, Ohio.

187. Personal communication Dr. Smith, Occupational Safety and Health Administration, Cincinnati, Ohio, November 1975.

188. U.S.E.P.A., Compilation of Air Pollution Emission Factors, RTP, North Carolina, 1973.

189. T. M. Barnes, Evaluation of Process Alternatives to Improve Control of Air Pollution from the Production of Coke.

190. P. H. Howard and P. R. Durkin, "Benzene Environmental Sources of Contamination, Ambient Levels and Fate," Syracuse University New York, December 1974.

191. D. Mackay and A.W. Wolkoff, "Rate of evaporation of low-solubility contaminants from water bodies to atmosphere," Environ. Sci. Technol., 7, 7, 611-614, 1973.

192. Office of the Federal Register, General Services Administration, Title 40-Protection of Environment, App. B.

193. U.S. Environmental Protection Agency, Compilation of Air Pollutant Emission Factors (Second Edition), Publication No. AP-42.

194. Daniel, W.A. and Heuss, J.M., "Ambient Air Quality and Automotive Emission Control," Journal of the Air Pollution Control Association, Vol. 24, Number 9, September 1974.

195. Personal communication, B. Baratz, The MITRE Corporation, December 1975.